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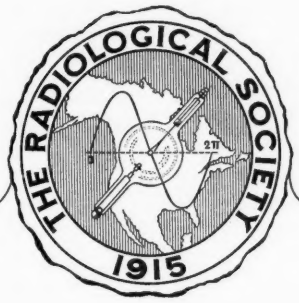

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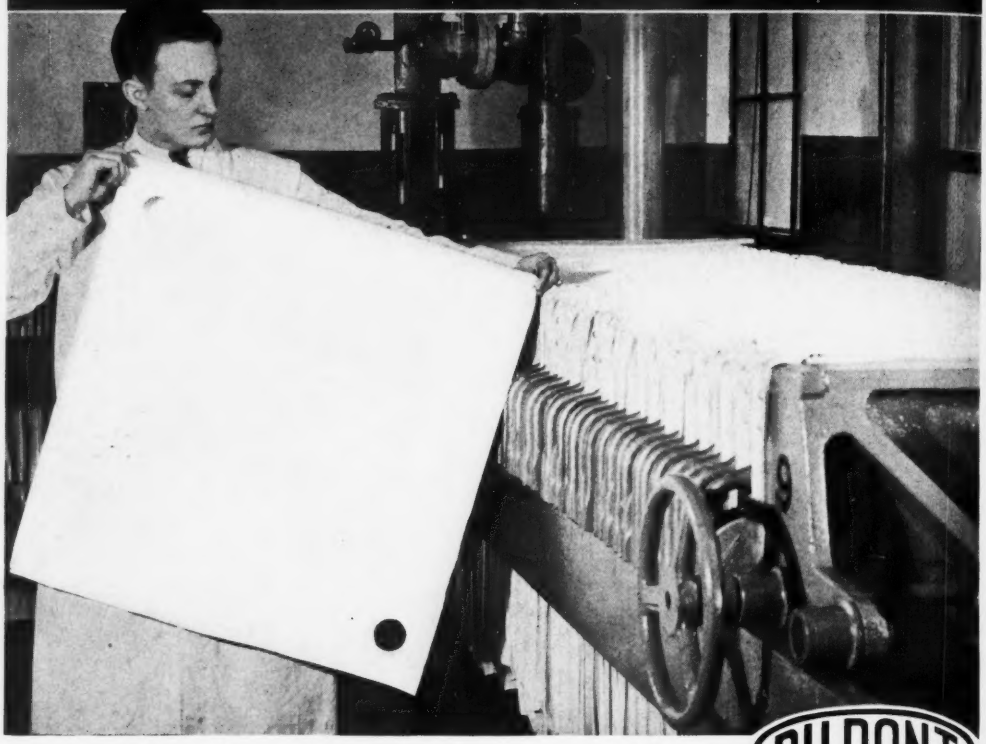

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VOL. 26 JANUARY, 1936 NO. 1

THE PRESENT MODE IN DEEP X-RAY THERAPY (COUTARD)[1]

By FREDERICK W. O'BRIEN, A.B., M.D., *Boston*

THE history of therapy by the medium of x-rays has been evolutionary by the very nature of things. The discovery of the x-rays occurred within the memory of most of us; electrical circuits and tubes have been developed for their production with amazing skill by our modern technological craftsmen. Their application in the therapeusis of malignant disease has been carried on from the start with zeal and fruitfulness, encompassed, however, by the limitations of any biologic problem in the human sphere. It is one thing to chart a dose by mathematical formulæ and quite another thing to apply it on the living subject.

Roentgen dermatitis was the lode star that showed the way in radiation therapy, but, to change the figure, it was in reality a mill stone, for, despite our newly acquired instruments of precision that measure the quantity and quality of x-rays, the total dose that can be safely administered continues to remain delimited by the only biologic norm that we have, namely, the skin erythema.

It was soon learned that an x-ray beam was heterogeneous, but that, by the introduction of suitable filters, undesirable skin effects could be avoided or their appearance deferred.

The shorter and more penetrating x-ray wave lengths are found in the higher voltages, which fact partly explains the modern race among manufacturers to produce energizing apparatus of capacities unheard of before our time. The gamma radiation of radium is very much shorter than the shortest x-ray wave length produced by our everyday working high voltage x-ray machines. However, there is no definite evidence that wave lengths *per se* have any specific effect. There is no biologic evidence that the tissue effect is any different when using a four-gram radium pack or 200 kv. under comparable conditions. This is not to say that x-rays and radium are interchangeable in our armamentarium, that one cancels the other—not at all. One is complementary to the other. What I wish to stress is that the radiation effect appears to depend entirely upon the amount of radiation absorbed, not on its wave length.

An x-ray dose is a very relative thing—talked about glibly since the introduction of the r meter, an instrument which measures the amount of x-rays ionized under given conditions. This quality is called an r unit, and is the product of the amount of a cross-sectional arc of a beam of x-rays reaching a point in a unit of time. The total number of r units prescribed or administered is only part of the story and far from informative. Physical set-ups and the time limits of the total dose are essential items of information; the wave length of the beam, the size of the skin

[1] Read in part at the Mid-winter meeting of the New England Roentgen Ray Society, Boston City Hospital, Jan. 25, 1935, and the Academy of Physical Medicine, Atlantic City, June 12, 1935.

portal, and the location of the measuring instrument are integral. No matter the number of r units, the skin effect is the ultimate arbiter, and, as we shall see, skin effect and the number of r units have a chronological relationship of major importance.

Back of all our effort has been the thought of a lethal dose, or that amount necessary to sterilize a neoplasm. There is no question but that the death of the tumor cell may be a direct effect of external radiation in superficial and some deep-seated lesions, although more often in the latter the effect has been an indirect one.

Two schools of practice have been current, partly dependent upon the concept of their followers as to how irradiation acts. One attempts to obtain a maximum tissue effect by a single or at most a few exposures to high voltage radiation, and the other by divided doses to obtain a cumulative effect. A variation of the latter, known as the saturation method, is an attempt to maintain by the addition of smaller doses a biologic effect produced by an initial large dose. Each of the smaller doses is presumed to produce an effect equal to that lost by recovery of the tissues.

The method of the moment which has attracted very wide and apparently deserved attention is a divided dose method, as practised by Henri Coutard, of the Radium Institute of the University of Paris. It has been succinctly described as the protracted, fractional high voltage low intensity method.

When Coutard first came to this country in 1931, he reported a series (1) of epitheliomas of the tonsillar region, hypopharynx, and larynx, treated by this method from 1920 to 1926. More recently in Zürich, at the Fourth International Congress of Radiology, he reported results with this method from 1926 to 1933, and summarized his work of about fifteen years, which was most stimulating.

The method essentially is as follows: high voltage x-rays (190–200 kv.), 3–4 ma. of current filtered by 2 mm. zinc and 3 mm. of aluminum; a focal skin distance of 50–60 cm. for lesions of the head and neck, and 80–100 cm. for deeply seated lesions.

Each dose is protracted; for example, 150 to 180 r are given in from 50 to 60 minutes. This low intensity of about 3 r per minute is directly proportional to the voltage, current, filtration, distance, and size of portal. The quantity of energy delivered is measured on the skin, which fact must always be kept clearly in mind, since common practice in this country is to report r units as measured in air without back-scattering.

Each dose is highly fractionated. When treating two opposite portals, as in laryngeal cancer, Coutard employs a time dose of one hour in the forenoon and one-half hour in the afternoon with treatments daily.

Small portals are used preferably, with a chronological distribution of dosage over a period of from 14 to 90 days, so that a high total dosage in r units is piled up—some ten to twenty or more times the total that could be given safely with the old methods. The total dose is estimated and controlled by the kind of tumor, its location, whether or not metastasis is present, and the general condition of the patient.

The startling thing about his demonstration to the uninitiated, in which class most of us were when he first came to this country, was that he produced a radiodermatitis of a second degree and a radioepithelitis of the mucous membranes which, while not without discomfort to the patient (some needing an emergency tracheotomy), went on to repair under the proper conditions without appreciable damage and with definite benefit to the patient.

The cutaneous reaction appeared about the fourteenth day, presenting loss of the epithelial layers and denudation of the dermis, with exudation. The time of the appearance of the epithelitis is dependent upon the site of the lesion in the larynx, and consists of denudation of the corion, which becomes covered by fibrin. It is to be noted that these reactions became less

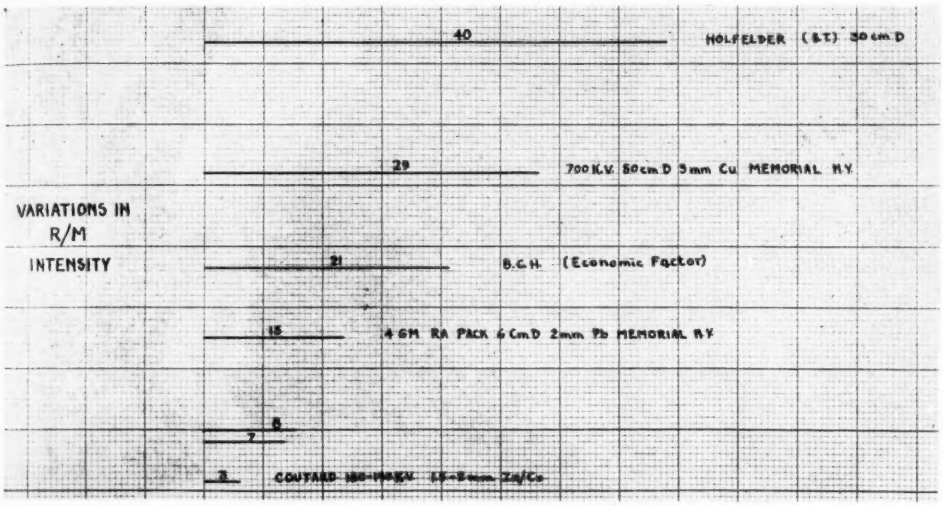

Fig. 1. Comparative r per minute intensities.

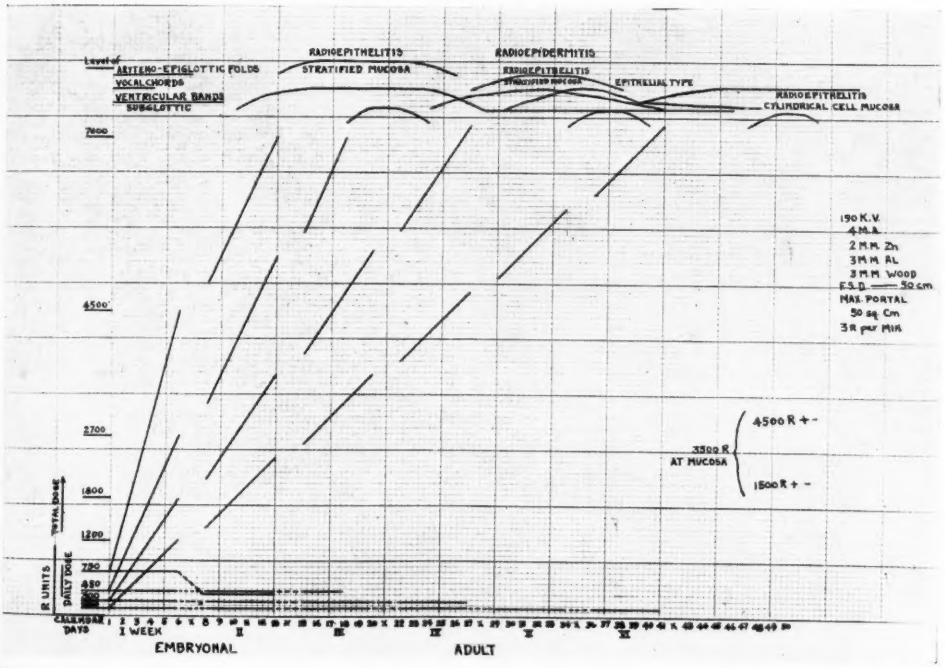

Fig. 2. Graphic analysis of Coutard's method, illustrating time spread out as total r per day dose is decreased.

severe, as in the perfection of his technic Coutard modified his total daily r dose.

From 1920 to 1926, Coutard attempted to get in a total dose of 7,000 r within approximately two weeks. During this time he noted the most favorable effect only on tumors of the embryonic type. When he increased the total doses to 10,000 and 12,000 r within the same time limit, adult type tumors remained uninfluenced.

From 1926 on he began to modify the total r dose per day and dropped from 750

Fig. 3.

Fig. 4.

Fig. 3. Refractory rodent ulcer of ten years' duration, treated with x-rays (old methods) and radium. Before treatment with Coutard method.
Fig. 4. Same lesion as shown in Figure 3, after treatment with Coutard method.

r to 450 to 300 and then to 200 r and even less, with a consequent chronological spreadout. By this procedure he began to obtain cures of adult type carcinoma.

His philosophy in explanation of this is well thought out and will be found in detail in his latest English publication (2) from which the following statements have been taken.

He believes that the nutrition of the cells of an epithelioma during irradiation is dependent upon the conservation of the medium in which it lies, namely, the vasculo-connective tissue. If the total or daily dose, or the intensity per minute, has been too high, or the field has been too large in relation to the dose, or if the filtration or the tension has been too low, the connective tissue may be modified and thereby the nutrition of the epithelioma cells may become difficult.

The cellular radiosensitivity, which seems linked up not only with the usefulness of the cells but, above all, with the activity of their interchanges with the vasculo-connective tissue, is modified—often diminished and sometimes depressed. The cancer cells behave clinically as if they had become insensitive to the irradiation: this is what Regaud described in 1913 under the name of radio-vaccination.

In the case of a very embryonic tumor, the radiomodifications of the vasculo-connective tissue have a very slight effect upon the radiosensitivity of the cancer cells because this sensitivity is pronounced. In a highly differentiated tumor, the radiomodifications of the vasculo-connective tissue are of the utmost importance because the radiosensitivity of these tumors is feeble.

In the treatment of highly embryonic, very radiosensitive cancers, the energy factor seems to be a direct and rapid one, the cells dying very soon and generally disappearing toward the twenty-fifth day. The vasculo-connective tissue seems to play only a very small part and is more often not modified after the destruction of the cancer cells.

In the embryonic cancers, a good chronologic distribution seems to insure protection to the surrounding tissue. As regards the differentiated cancers, the chronology is a different character—on the one hand, the accidents to the general tissues are exceptional on account of the long time over which the treatment is spread, and

on the other hand, disappearance of the cancer cells does not happen by cellular destruction but by a sort of late cellular evolution similar to the normal evolution. Between the forty-fifth and ninetieth days, the neoplastic masses, which have generally diminished in volume during the previous irradiations, increase anew for from eight to ten days, giving the impression of a rapidly growing recurrence; they then cover themselves with "horny" formations or glandular secretions and diminish anew inside and little by little disappear. Here, he says, the chronology is no longer protective—it seems to be evolutionary.

Criticism of the method has been made from several points of view, to the fore being naturally the very long time and the expenditure required. If his method is followed literally, each patient needing from 30 to 60 hours of radiation, it is obvious that the tube expenditure must be high. It has been estimated on the Continent that the hospital cost of each patient is something like three hundred dollars. The time required is consequential to the low intensity he employs (Fig. 1). It is obvious that if we must follow his method according to the letter, our therapy departments will have to be greatly enlarged.

As it happens in the metropolitan centers in this country, we must treat ten patients to his one. Since the appearance of his work radiation therapists here, because of the amount of clinical material that must be cleared through their departments, have modified his technic to the extent of increasing the r per minute intensity, thereby shortening the treatment time per séance in some cases from one hour to ten minutes. It remains to be seen whether or not our economic procedures are justified. I think they may be, as I analyze his published work.

From the graph it is evident that he obtained his cures of adult type carcinoma of the larynx when he cut down his total r unit daily dose without any change in intensity (Fig. 2). From this it is logical to conclude that, limiting the total daily dose with consequent chronologic spread-out to obtain a high total dose in r units was paramount. Coutard's insistence on low intensity is partly founded on analogy drawn from the low intensity radium method employed in cervical cancer at the Curie Institute. Practically it has been the experience in this country that if a daily dose not exceeding 200 r is used, irrespective of the r per minute intensity, the clinical results are favorable and comparable with his method when followed strictly.

Coutard's method, modified or otherwise, is now being used in carcinoma of the esophagus (3), cancer of the breast, uterus, and stomach. Brilliant results have been obtained in laryngeal cancer (4) and refractory rodent ulcer (Figs. 3 and 4). In these lesions the opportunity to deliver a depth dose with more uniform distribution and greater absorption presents itself. In other locations the problem of depth dose is more complicated, and for final evaluation of the method we must wait.

465 Beacon St.

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BENIGN TUMORS OF THE STOMACH[1]

OBSERVATIONS ON THEIR INCIDENCE AND MALIGNANT DEGENERATION

By LEO G. RIGLER, M.D., *Minneapolis, Minn., and* LESTER G. ERICKSEN, M.D., *Dubuque, Iowa*

From the Department of Radiology of the University of Minnesota and the University Hospital, Minneapolis

THE microscopic distinction between benign and malignant neoplasms of the stomach is occasionally impossible, often hazy, and, even in those instances in which it is clear-cut, there is a strong possibility that a lesion appearing benign may eventually become malignant. It would seem, therefore, almost foolhardy to attempt to distinguish them roentgenologically were it not for our experience which indicates that frequently, at least, even this differentiation may be made. The roentgenologic diagnosis "benign gastric tumor" has been confirmed frequently enough so that this terminology seems eminently justified. The demonstration by roentgen examination of the stomach of a benign tumor no larger than a hemp seed (2), or of a carcinoma which is barely distinguishable macroscopically (20), gives an indication of the possibilities of this method and lends encouragement to attempts at making such fine distinctions.

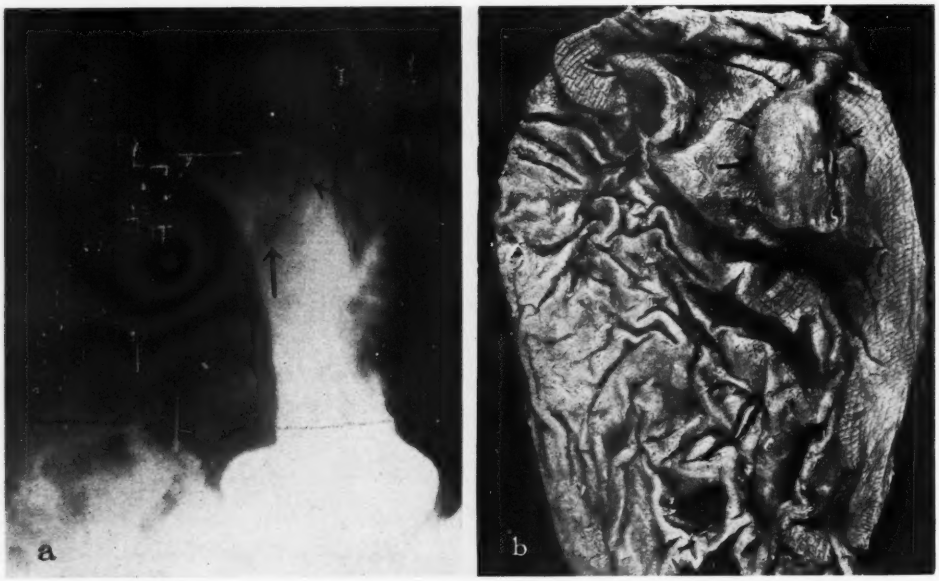

Fig. 1. Case of benign polyp, showing close correspondence between roentgenographic and autopsy findings. (*A*) Roentgenogram showing kidney-shaped defect near lesser curvature in cardiac end (arrows). (*B*) Photograph of postmortem specimen of stomach, showing pedunculated tumor (arrows) corresponding exactly to defect in barium outline.

An apt illustration of the faithfulness with which the roentgenogram may reproduce the gross pathology of stomach tumors is shown in Figure 1. The film (Fig. 1-*A*) shows a small kidney-shaped defect in the cardiac end near the lesser curvature,

[1] Presented as an exhibit at the Annual Meeting of the Radiological Society of North America, in Memphis, Tenn., Dec. 3–7, 1934.

which is sharply defined, and shows no evidence of infiltration of the gastric wall. The photograph of the opened stomach removed at autopsy is shown in Figure 1-*B*. The typical appearance of a pedunculated benign polyp is shown, corresponding most exactly with the filling defect demonstrated in the roentgenogram.

The symptomatology, diagnostic signs, and pathology of benign tumors of the stomach have been repeatedly amplified in excellent papers on this subject during the past ten years (1, 4, 6, 7, 10, 12, 15, 16, 19, 21), and we do not propose here to review these phases of this subject. We wish merely to present some evidence of the actual incidence of benign gastric tumors, and to call attention again to the frequency with which they undergo malignant degeneration.

When one of us (L. G. R.) reported 15 cases in 1930 (19), there were already in the literature records of almost a thousand and from one clinic alone some fifty cases had been described (6). Nevertheless, non-malignant tumors of the stomach are still considered relatively rare, and they obtain little attention in the ordinary text-book.

Data as to the incidence of benign gastric tumors are variable. Eusterman and Senty (8), in 1922, reported that 1.3 per cent of all the tumors of the stomach were benign. Kirklin and Weber (11) give the incidence as less than 2 per cent, while Dwyer (5) found only three cases in over five thousand gastro-intestinal examinations, the ratio to malignant tumors being 1 to 39. Lockwood (12) reported an incidence of about 4.3 per cent, and Eusterman, in discussing this paper, indicated his conviction that the incidence is higher than has been generally believed.

It seems reasonable to suppose that the actual incidence of benign gastric tumors should be greater than that reported on the basis of clinical, roentgenologic, or sugical data. Many of these lesions give no symptoms or signs and, therefore, are not observed; many are no doubt overlooked even on roentgen examination. Only those which are thought to be malignant or which produce marked symptoms are likely to come to operation. For these reasons the frequency as revealed by postmortem studies, naturally, would be much greater. There is one corrective factor in this regard, namely, that many of these tumors tend to become malignant and by the time autopsy is done they may be classified with the carcinomas, although originaliy they were simple polypi.

In 1930 (19) one of us (L. G. R.) reported a rather high incidence of benign lesions in a small series of cases of gastric tumors. With a view to determining more accurately what this ratio actually is, we undertook a study of the various lesions in this region uncovered by roentgen examination over a five-year period in the University of Minnesota Hospitals.

TABLE I.—INCIDENCE OF GASTRO-DUODENAL LESIONS FOUND DURING A FIVE-YEAR PERIOD BY ROENTGEN EXAMINATION OF PATIENTS WITH GASTRO-INTESTINAL COMPLAINTS

Total number of cases examined		4,236	
Total cases with gastro-duodenal lesions		1,547	36.3%

Diagnosis	No.	Per cent
Carcinoma of stomach	208	13.4
Malignancy of stomach, miscellaneous	4	0.3
Benign tumors of stomach	25	1.6
Benign tumors of duodenum	2	0.1
Gastric ulcer, benign	129	8.3
Duodenal ulcer	838	54.2
With definite niche 186		
With obstruction 102		
Gastric and duodenal ulcer	15	1.0
Gastro-jejunal ulcer	12	0.8
Gastric diverticulum	2	0.1
Duodenal diverticulum	58	3.7
Para-esophageal hernia	44	2.8
Hypertrophy of gastric rugæ—chronic hypertrophic gastritis(?)	61	4.0
Periduodenal adhesions	46	3.0
Extrinsic pressure	37	2.3
Miscellaneous	10	0.7
Doubtful and ill-defined findings	56	3.6

There is listed in Table I the roentgen diagnosis in 4,236 patients referred for examination because of gastro-intestinal complaints. Somewhat more than a third of these showed definite evidences of abnormality of the stomach and duodenum. The incidence of all the types of important pathology is presented in order to indicate, insofar as is possible, the class of material upon which this study is based.

Certain outstanding characteristics of this group of cases may be noted. The ratio of gastric to duodenal ulcer (1 to 6.5) is somewhat higher than that usually reported, and may be compared to the figures published by Percy and Beilin (17). The ratio of gastro-duodenal tumors to gastric ulcer is about 2 to 1, and to duodenal ulcer about 2 to 6.5. The comparatively high incidence of para-esophageal hernia (1 per cent of all the cases examined) reflects a particular interest in this lesion.

TABLE II.—RELATIVE INCIDENCE OF GASTRO-DUODENAL TUMORS FOUND BY ROENTGEN EXAMINATION

			Per cent
Total number of tumors of the stomach		239	
Malignant tumors of the stomach		212	88.7
Carcinoma	208		
Neurosarcoma	2		
Leukemia	1		
Lymphosarcoma	1		
Benign tumors of the stomach		21	8.8
Benign tumors of the stomach with malignant degeneration		4	1.7
Benign tumors of duodenum		2	0.8

Malignant tumors of the stomach were found nine times as frequently as the benign type in this series of roentgen examinations.

There were 239 patients with tumors of the stomach and duodenum. The analysis of these tumors and their relative incidence is shown in Table II. After eliminating from the benign group every doubtful case, there were 27 cases which we feel confident had non-malignant tumors. Of the patients diagnosed as having benign tumors, ten have been explored surgically or have come to autopsy. In one case the tumor proved to be a neurosarcoma; in another, the tumor showed malignant degeneration, but otherwise the findings have been proved to be accurate. Assuming the substantial correctness of these diagnoses, the benign lesions represent approximately 11 per cent of all the tumors observed. This incidence is strikingly different from that found by other workers but tends to confirm the high incidence previously reported (19) by one of us (L. G. R.).

It should be pointed out that this is a small series, so that the actual figure of 11 per cent probably is not at all accurate. Furthermore, the percentage of error which necessarily attends the roentgen examination of the stomach both as to benign and malignant lesions will further militate against the accuracy of this figure. On the other hand, as stated above, it is very probable that the incidence of benign tumors, roentgenologically observed, is distinctly lower than the actual incidence; many patients who have such tumors do not even appear for roentgen examination because the tumor produces no symptoms. The figure of 11 per cent, therefore, while not in any sense statistically correct, is in all probability low rather than high.

TABLE III.—INCIDENCE OF GASTRO-DUODENAL LESIONS FOUND BY PATHOLOGIC EXAMINATION

Total number of cases examined	6,242
Total cases with gastro-duodenal lesions	392

Diagnosis	No.	Per cent
Carcinoma of stomach	130	33.2
Malignant tumors of stomach, miscellaneous	8	2.0
Benign tumors of stomach	49	12.5
Benign tumors of duodenum	5	1.5
Carcinoma of duodenum and papilla	2	0.5
Gastric ulcer, benign	72	18.3
Duodenal ulcer	95	24.2
Gastro-jejunal ulcer	3	0.8
Gastric diverticulum	1	0.3
Duodenal diverticulum	15	3.8
Para-esophageal hernia	2	0.5
Miscellaneous	10	2.6

In order to determine whether the discrepancy between our data and that reported by others was due to our own overenthusiasm for this diagnosis, we investigated the records of the Department of Pathology of the University of Minnesota with regard to autopsies alone. All the data on postmortem examinations for a period of four years were examined chronologically, the diagnosis in 6,242 cases being tabulated (Table III). Of these, there were gastro-duodenal lesions in 392 cases. It should be borne in mind in this connection that the much higher incidence of abnormality in this region observed on roentgen examination is due to the selective character of these cases—patients with gastro-intestinal symptoms. Furthermore,

it should be noted that the postmortem studies include numerous still-births, deaths in early infancy, and accidental deaths in which the incidence of gastro-duodenal lesions is naturally very low.

Certain criticisms of this type of study must be entertained. The autopsies are routine procedures and were not made with any particular effort to detect lesions of the gastro-intestinal tract. It is probable that in some cases, particularly those in which the cause of death was readily apparent and due to some cause outside the stomach or duodenum, the latter were examined rather superficially and lesions might have been overlooked. The autopsies were done by a large number of different pathologists. In spite of these necessarily inevitable, albeit regrettable sources of error, the incidence of lesions may be considered to be roughly correct for such a group of patients. These are predominantly male, of a rather advanced age, and generally of a low social class.

As is commonly the case in postmortem studies, the incidence of gastric ulcer compared to duodenal ulcer is relatively high, while the number of tumors is much greater than either. It may be noted that duodenal ulcer represents only 24 per cent of the cases in which lesions were found, while in the living patients this diagnosis occurs in 54 per cent of the cases. On the other hand, gastric ulcer is present in 18 per cent of the autopsied cases and in only 8 per cent of the clinical group. A striking difference is shown in the para-esophageal hernia diagnoses, the percentage on roentgen examination being over five times that found at autopsy. This may be best explained, we believe, by the failure of the pathologist either to search for or to detect the smaller dilatations of the hiatus esophagus.

There were, in this group, 194 cases with tumors of the stomach and duodenum. An analysis of these is presented in Table IV, and produces the astonishing fact that over 25 per cent of all the tumors of the stomach and duodenum are benign, even if the benign lesions with malignant degeneration are not included. Malignant tumors of the stomach are shown to be only three times as frequent as the benign variety. It should be noted here that these benign tumors do not include the extremely frequent, tiny, but clinically insignificant leiomyomas reported by Rieniets (18). The fact is that any error in these figures is likely to militate against an increased frequency of the benign lesions rather than in favor of it. The average pathologist is inclined to consider a stomach tumor malignant until it is proved otherwise. Furthermore, he is much more likely to overlook a small polyp of the stomach when making a superficial examination than he would a carcinoma. On the other hand, it is possible that a more intensive histologic study, such as was made by McRoberts (13), of some of the lesions considered to be benign might have proved them otherwise. For practical purposes, however, this need not be considered too strongly. A tumor which cannot be readily detected as malignant by ordinary gross and microscopic examinations would most certainly have to be classified as a benign lesion clinically and roentgenologically.

At first glance, the incidence here reported may appear incredibly high. We do not believe that our cases are sufficient in number or well enough controlled to give absolutely accurate data on the frequency of non-malignant tumors of the stomach.

TABLE IV.—RELATIVE INCIDENCE OF GASTRO-DUODENAL TUMORS FOUND ON PATHOLOGIC EXAMINATION

Total cases with gastro-duodenal tumors 194

Diagnosis		No.	Per cent
Malignant tumors of stomach		138	71.1
Carcinoma	130		
Sarcoma	3		
Leukemia	4		
Hodgkin's disease	1		
Benign tumors of stomach		45	23.2
Polyp	31		
Leiomyoma	12		
Fibroma	2		
Benign tumors of stomach with malignant degeneration		4	2.1
Carcinoma of duodenum		2	1.0
Benign papilloma of duodenum		5	2.6

Malignant tumors of the stomach are only three times as frequent as the benign type in this series of autopsies and surgical specimens.

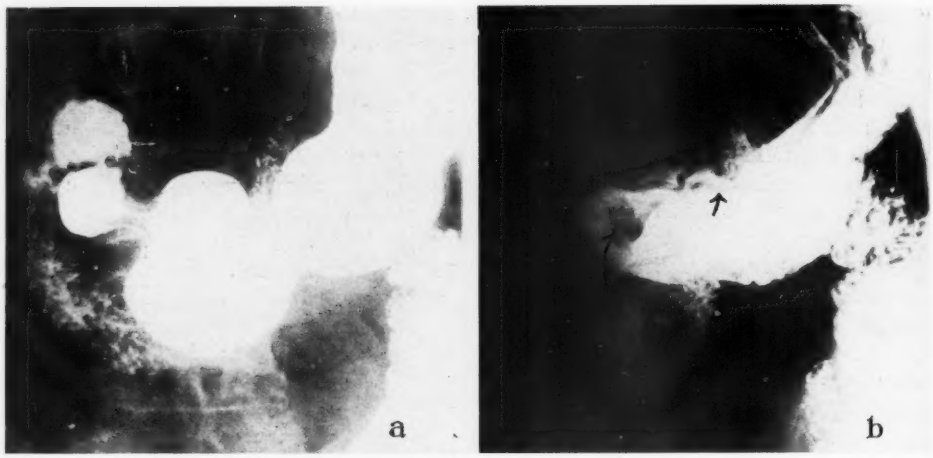

Fig. 2. Case of benign tumor, showing importance of technic. (*A*) Ordinary roentgenogram of stomach, overfilled and without pressure. Note normal appearance of pyloric end and passage of peristalsis through it. (*B*) Roentgenogram of same case, with underfilling and pressure over antrum. Note typical defect of benign tumor in pyloric third (lower arrow) and appearance of mucous membrane fold (upper arrow).

The series is large enough, however, to indicate beyond any doubt that the low incidence (1 to 4.5 per cent) reported by other observers is grossly in error. Our opinion in that regard is confirmed by the work of Stewart (21), who has reported figures in the same category derived from postmortem studies. In 11,000 cases, he found 47 instances of benign and 263 of malignant neoplasms of the stomach, indicating that over 15 per cent of the stomach tumors were benign. The work of Rieniets (18) is also significant in this regard. In 200 consecutive autopsies, he found 12 stomachs with carcinoma and 32 stomachs with one or more leiomyomas. While most of these were exceedingly small and would escape detection in the routine postmortem examination, there were a number, the diameter of which was almost one centimeter. His findings suggest that more careful examination, even at autopsy, would reveal a greater number of benign tumors than has hitherto been reported.

The frequency with which benign tumors of the stomach are found at autopsy serves to confirm our roentgenologic studies. There are numerous factors which explain the failure to discover more of these tumors by the ordinary roentgen examination of the gastro-intestinal tract. As Moore (15) so well pointed out, familiarity with any particular lesion is an important factor. It is no doubt true that many benign tumors of the stomach are symptomless, hence there is no opportunity presented for detecting them; many of them, when found, are thought to be malignant, and many are overlooked altogether, even on roentgen examination.

The most important factor, we believe, is concerned with the technic of examination of the stomach. As was pointed out in a previous publication (19), overfilling of the stomach will readily obliterate the evidences of these tumors. It is necessary in many instances to produce pressure on the stomach, films being made during roentgenoscopy for better demonstration of lesions, brought out, by proper manipulation, during the screen examinations. Heavy penetration of the stomach when films are made is of considerable importance, as small defects from tumors of the anterior or posterior wall may thus be brought out.

The vital importance of pressure and under-filling of the stomach is illustrated in Figure 2. The roentgenogram of the stomach (Fig. 2-*A*) made in the routine

fashion shows no defect or any other abnormality. A film (Fig. 2-*B*), made at about the same time with pressure and with the barium shadow when films of average penetration were made.

The various roentgen signs of benign

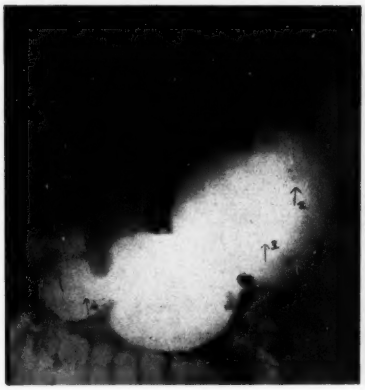

Fig. 3

Fig. 4.

Fig. 3. Case of multiple benign polypi. Note numerous defects in the stomach and one at base of duodenal bulb, due to prolapse of tumor through the pylorus. The defects shown by arrows 1, 2, 3, and 4 could not be seen in underpenetrated films, and are made out even here with difficulty because the tumors are so small in relation to the lumen of the stomach.

Fig. 4. Benign polyp of middle third of stomach associated with moderate hypertrophy of the gastric mucosa. The distinction of the tumor defect (upper arrow) from the defect produced by a fold of mucous membrane (lower arrow) is made with difficulty. Examination of numerous films in various positions to demonstrate the independence of the tumor defect from the mucosal pattern must be done. Note the brilliant manner in which the pressure bag brings out the mucosal pattern and the tumor defect as contrasted with the homogeneous appearance of the distal portion of the stomach.

less barium, shows the characteristic somewhat rounded, sharply demarcated filling defect of a benign tumor. By roentgenoscopy, this defect appeared to be somewhat mobile; the walls of the stomach about it were seen to be flexible and, as also shown in Fig. 2-*A*, there was no interference with peristalsis. We have had numerous cases of this type, which emphasize how readily these small, mobile tumors may be overlooked.

The importance of over-penetration in the production of the roentgenogram in these cases is well illustrated in Figure 3. This is a case with multiple polypi, one of which may be noted to have prolapsed into the duodenum. Only with a heavy exposure could the small, rather flat tumors near the greater curvature (arrows numbered 1, 2, 3, 4) be brought out. The anteroposterior diameter of these tumors was insufficient to produce any change in

tumors of the stomach have been fully discussed in other publications (7, 9, 10, 12, 15, 19). Likewise, the factors in differential diagnosis have been elaborated by these authors. Certain points of differentiation should be emphasized. The distinction from food particles is relatively easy and can almost always be made by repeated examination. The extreme mobility of foreign bodies in the stomach and the inconstancy of the defects which they produce may readily rule them out.

The differentiation of benign tumors from hypertrophied folds of mucous membrane presents a much more difficult problem. In cases of chronic hypertrophic gastritis, large folds seen on end may simulate a small polyp very exactly. The distinction may be made by tracing the defect into the mucosal pattern, while in the tumor the mucous membrane shadows are completely obliterated. The extension of the folds

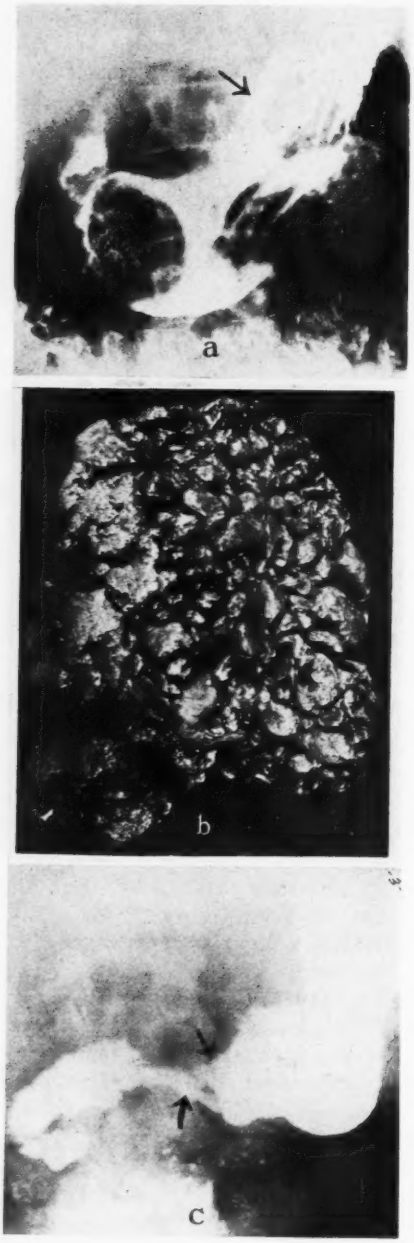

Fig. 5. Case of benign papillomas of stomach with malignant degeneration. (*A*) Roentgenogram with pressure, showing two large papillomas (arrows). Note the extension of barium among the papillary folds in the lower defect, giving an indication that it is that type of tumor. Note also mucous membrane pattern on greater curvature. (*B*)

(*See next column*)

Fig. 6. Photograph of postmortem specimen of stomach, showing benign polyp (left) and polypoid carcinoma (right) side by side (microscopically proved).

around but not into the area of a benign tumor is well shown in Figure 2-*A*. Figure 4 illustrates a small benign polyp, differentiated with difficulty from the folds of mucous membrane around it. In some instances, we believe, the distinction will be virtually impossible if the tumor is very small. Most cases of gastritis, however, will show a multiplicity of ridge-like defects which are tortuous, long and narrow, rather than round, and recognizable as rugæ of large size.

Carcinoma in most instances will be readily recognized by the evidences of deep infiltration into the wall of the stomach. Absence of peristalsis through the involved area, rigidity on manipulation with the palpating finger, immobility, irregularity, lack of roundness, lack of sharp demarcation of the defect, and reduction of the lumen of the stomach all characterize a malignant lesion. Nevertheless, it must be borne in mind that certain carcinomas do not infiltrate to any appreciable extent below the mucosa, and may, therefore, fail to produce many of these signs. Likewise, certain benign lesions are sufficiently submucosal to

Photograph of one of the excised specimens; microscopically, it is apparently benign. Note correspondence with the roentgen findings in Figure 2-*A*. (*C*) Roentgenogram four years later, showing extensive carcinoma at site of lower lesion.

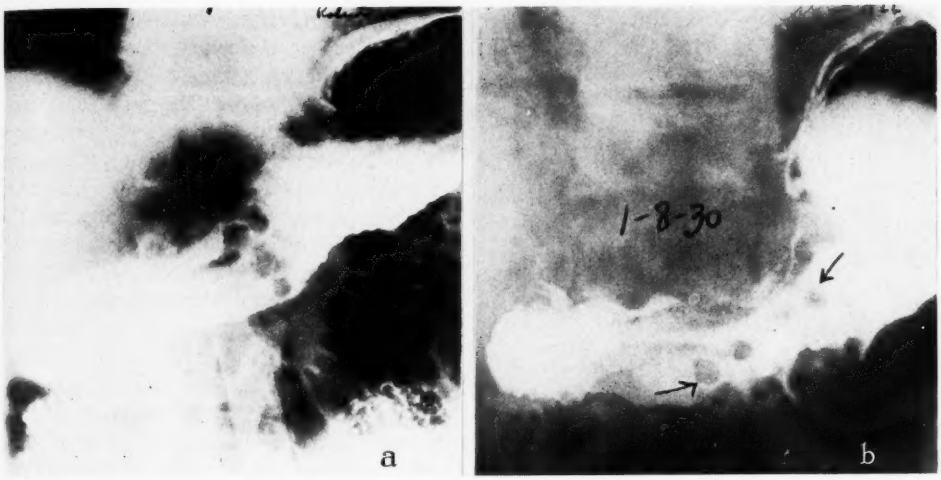

Fig. 7. Case of benign polypi of stomach with malignant degeneration. (*A*) Roentgenogram made with pressure on first examination. A number of rounded filling defects in the middle third of the stomach are shown. Peristalsis passed through this area unhindered. The tumors were movable and the gastric walls flexible. (*B*) Same case eight months later. Roentgenogram shows benign tumors and also characteristic carcinomatous infiltration of greater curvature. Proved at operation.

suggest that the muscle layers have been infiltrated. The differentiation of a benign from a malignant lesion in these unusual cases may be exceedingly difficult or impossible. As stated in the opening paragraph, this differentiation may be impossible even at autopsy.

The importance of the early diagnosis of benign tumors of the stomach, particularly those of epithelial origin, is twofold. They may be a cause of numerous symptoms which can readily be relieved by adequate surgery. Possibly more important is the fact that they frequently degenerate and become carcinomatous. That benign polypi in the rectum and colon are a common source of carcinoma has long been recognized, and the same process takes place elsewhere in the body. It has not been so generally realized, perhaps, that gastric carcinomas may arise from benign epithelial tumors. Stewart (21), in a study of 263 cases of carcinoma of the stomach, found 13 associated with polypi—about 4.5 per cent. Of 47 cases of benign tumor, he found positive evidence in three of malignant degeneration; he considers them a definite precancerous lesion. Miller, Eliason, and Wright (14), out of 23 cases studied, found eight benign tumors degenerating into carcinomas. These represented about 4 per cent of 200 cases of carcinoma of the stomach. Benedict and Allen (3), Kirklin and Broders (10), and Brunn and Pearl (4) have reviewed the literature on this subject thoroughly. The concensus of opinion would indicate that from 12 to 60 per cent of benign polypi eventually become cancerous. McRoberts (13) found microscopic evidences of malignant degeneration in four out of five cases intensively studied. Balfour, in discussing this paper, expressed the belief of most students of this subject that benign tumors of the stomach should be treated as if they were cancers or, at least, precancerous lesions.

The case shown in Figure 5 illustrates this phase of the problem well. First seen in 1929, this patient presented the clinical findings of a benign tumor of the stomach. Roentgen examination (Fig. 5-*A*) demonstrated two benign papillomas which were surgically excised, but resection of the stomach was not done. The specimens obtained (Fig. 5-*B*) were typical benign papillomas which, on microscopic examination, showed no evidences of malignancy.

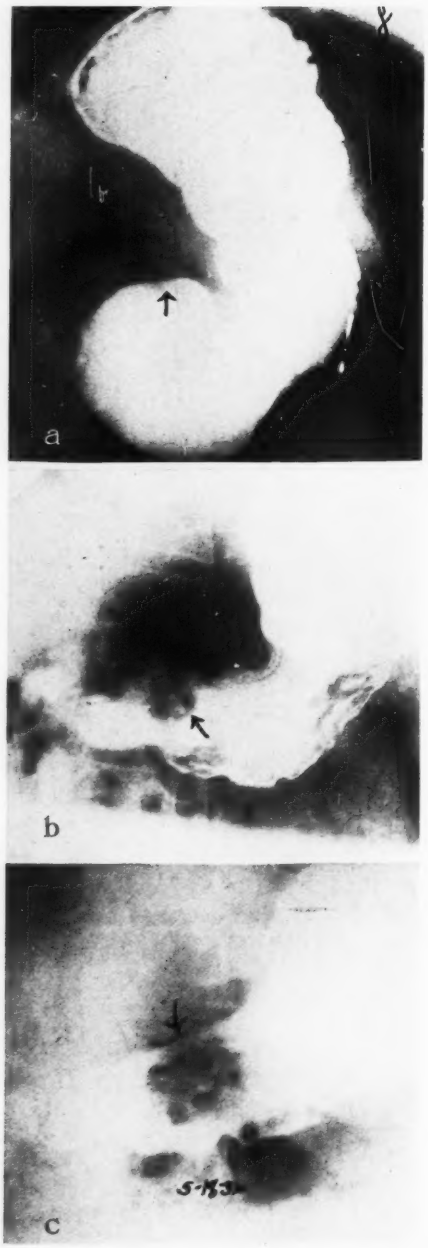

Fig. 8. Case of benign polyp of stomach, with recurrence and probably malignant degeneration. (*A*) Roentgenogram showing duodenal deformity and faint impression on lesser curvature of stomach (arrow). (*B*) Same case. Re-examination following day with under-filling and pressure. Note characteristic defect of a benign tumor on lesser curvature. This was excised and microscopically proved to be benign. (*C*) Same case 27 months later. Roentgenogram shows recurrence of tumor with infiltration of curvature, probably due to malignant degeneration.

(*See next column*)

Four years later, the patient returned with the classical symptoms of carcinoma of the stomach; this diagnosis was confirmed by roentgen examination (Fig. 5-*C*).

The association of benign and malignant lesions in close approximation has been repeatedly observed at autopsy. The photograph of a typical specimen is shown in Figure 6. Not only may the two lesions be distinguished from each other roentgenologically, in some cases, as previously reported (19), but the onset of malignant degeneration may be demonstrated as well. Such a case is illustrated in Figure 7. The patient presented himself with a fairly typical clinical and roentgen picture of multiple benign polypi. The first examination is shown in Figure 7-*A*. The sharply rounded defects seen in the middle third of the stomach were mobile, and peristalsis passed readily through this portion of the stomach. The patient refused operation; eight months later, he returned and the second examination (Fig. 7-*B*) revealed an irregular infiltrating lesion on the greater curvature, characteristic of carcinoma, together with the benign tumors previously observed. At operation, both types of tumor were found exactly as described.

Recurrence of these tumors after simple excision must also be expected in some instances. Such a case is illustrated in Figure 8. On the original examination (Fig. 8-*A*), the evidences of an old duodenal ulcer with marked deformity were found, together with a faint impression on the lesser curvature in the pyloric third (arrow). Re-examination the following day, with pressure and under-filling, revealed the typical benign polyp shown in Figure 8-*B*. This single tumor was found at operation and excised—it proved, on microscopic examination, to be benign. Eighteen months later, after a recurrence of symptoms, the patient was re-examined; a tumor on the greater curvature of small size was dis-

covered. Some nine months later, a further examination revealed a growth on the lesser curvature as well (Fig. 8-*C*), suggesting most probably that malignant degeneration had taken place in the recurrent benign lesion. Because of other complications, no further surgery was undertaken in this case, but the value of repeated examinations as revealing the change in conditions is well demonstrated.

On the other hand, we have had the opportunity of observing a number of patients over a period of years, particularly those with single lesions, without any change whatever in the appearance of the tumor. While realizing the importance of the extirpation of these tumors as a preventive of malignancy, it is occasionally difficult to recommend so serious an operation as gastric resection to a patient with only one small neoplasm. Under these circumstances, repeated roentgen examinations at frequent intervals are highly desirable as, in this way, the first onset of malignancy may possibly be detected.

With the increasing use of some type of mucosal technic in the roentgen examination of the gastro-intestinal tract, and with the increasing realization that benign tumors of the stomach are not rare, the roentgen diagnosis of this condition should become more and more frequent.

SUMMARY

In a series of 239 tumors of the stomach or duodenum, approximately 11 per cent were roentgenologically diagnosed as benign.

In a series of 194 tumors of the stomach or duodenum observed at autopsy, approximately 25 per cent were diagnosed as benign.

These groups of cases indicate a far greater frequency for non-malignant tumors of the stomach than has been previously reported.

The most important factor in the failure to recognize these cases roentgenologically is the technic of examination.

Many benign tumors of the stomach degenerate into carcinomas. This tendency is so marked that they should be treated as cancerous or precancerous lesions.

If radical surgery is not done, repeated roentgen examinations should be made at frequent intervals so that the onset of malignancy may be detected at the earliest possible moment.

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A STUDY OF CONES OR OTHER COLLIMATING DEVICES USED IN ROENTGEN THERAPY

By EDITH H. QUIMBY *and* L. D. MARINELLI, Department of Biophysics, Memorial Hospital, *New York City*

IN many roentgen therapy departments, open ports are being displaced by cones or other collimating devices, generally with two objects in view: the better assurance that the patient will not change his position during treatment, and the better control of the beam of radiation. About the first point there can be no question. When there is no mechanical control over the position of the patient, especially if the field is small, a slight displacement of the patient, sufficient to remove all or part of the diseased tissue from the field of radiation, may occur without its being noticed by the operator.

It is, however, not obvious what effect this attachment to the tube-holder may have upon the beam of radiation. It is certain that modifications in its quality and quantity will be brought about by the introduction of even a small amount of material into the path of the rays. The change in diaphragm system may also result in alterations in the beam. It is important to find out how great these changes may be, and what are their effects on the applicability of dosage data obtained with open ports. The present study was undertaken in order to obtain practical information along these lines.

The source of x-rays was a constant potential generator with air-cooled tube, operated for most of the work at 165 kv., but in a few experiments (which will be noted) at 200 kv. The primary filter was usually 0.54 mm. of copper. There is no reason to suppose that different results would have been obtained with any other set-up within the usual therapy range.

The attachments used were cylindrical, 30 cm. in length. They fitted into the base of the stationary tube-holder so that the distance from the target to the lower opening of the cylinder was always 65 cm. Each one was supplied with a removable top diaphragm which, when in place, limited the beam so that it would just fill the lower opening if the radiation came from a point source. When this diaphragm was removed, the top of the cylinder was directly open to the tube (except for the filter). Four cylinders were made of lead 3.2 mm. thick, having diameters of 5, 7, 8.8, and 15 cm., respectively. In addition, one was made of bakelite 3.2 mm. thick, and also 8.8 cm. in diameter.

For most of the work, the ionization chamber used was of the mesh type previously described (1), insuring the inclusion of the very soft secondary radiation in the measurements. In some cases a small spherical celluloid chamber was also employed. Readings were made with the Failla vacuum tube measuring instrument (2). The plan of the set-up is shown in Figure 1. With the bakelite cylinder it was necessary to use an extra lead diaphragm surrounding its base, as indicated in the inset drawing, to cut off radiation passing through the cylinder walls, which would fall on parts of the mesh chamber outside the regular beam. This diaphragm in no way interfered with the measurements, but served to keep data from the lead and bakelite cylinders comparable.

1. *Secondary Filter Necessary.*—The apparatus was adjusted so that the mouth of the cylinder was just outside the shielding lead case of the ionization chamber. No limiting top diaphragm was used, in order to have the maximum amount of scattered and secondary radiation from the walls of the cylinders. With the copper filter in place at the top, readings were taken for various secondary filters closing the bottom of the cylinders. Figure 2 shows the results for the 8.8 cm. lead cylinder. A single sheet of typewriter paper reduced the in-

tensity of the ionization from 100 to 88 per cent, and ten more sheets made no further difference. Thin sheets of aluminium, al-

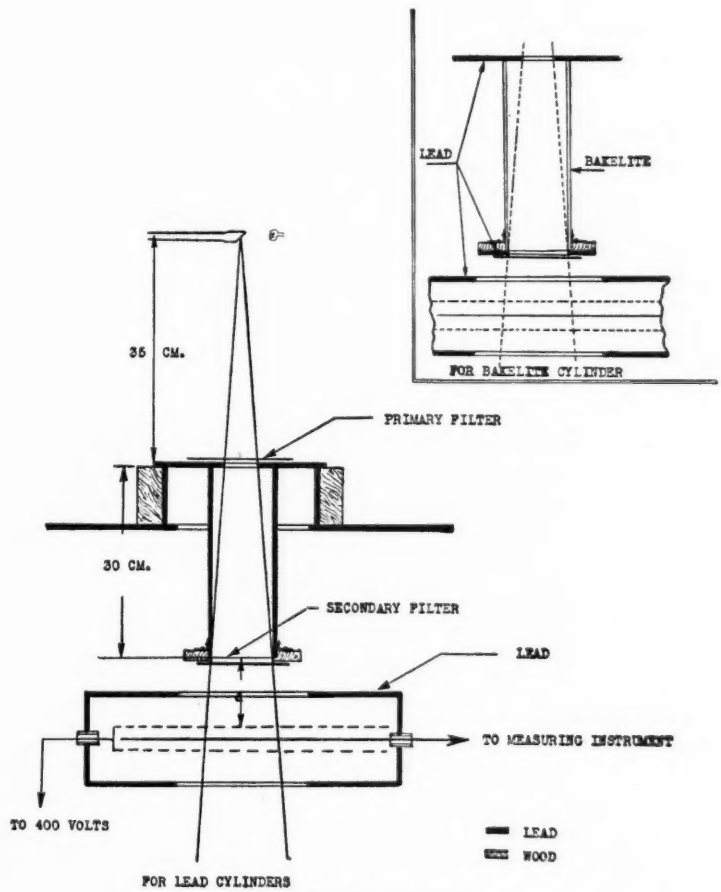

Fig. 1. Experimental set-up.

ways with one sheet of paper toward the ionization chamber, reduced the radiation as shown in Curve 2. After 0.3 mm. there was no appreciable decrease up to 1 mm. It is, therefore, concluded that 0.3 mm. aluminium plus one sheet of paper is a satisfactory secondary filter at the mouth of a lead cone. Instead of the paper, the aluminium could be covered with a thin layer of celluloid. If celluloid or bakelite is to be used as a substitute for the aluminium, as a secondary filter, it is evident from Curve 3 that about 1.5 mm. produce the required reduction in the radiation. The same results were obtained with all the lead cylinders, whether the limiting top diaphragm was in place or not. The results were the same on moving the ionization chamber up so that the mouth of the cylinder was only 1.5 cm. from the mesh; it was found that this filter was still sufficient.[1] With the bakelite cylinder, no secondary filter was necessary.

[1] The reason for not making all of these measurements with the cylinder as close as possible to the mesh was the difficulty of getting the filters in and out, with danger of damage to the chamber.

2. *Effect of Increasing Thickness of Bakelite or Other Organic Material at Bottom of Cone, Beyond Amount Required for* vestigating these effects, the type of ionization chamber used is of great importance. The mesh chamber, covering the whole

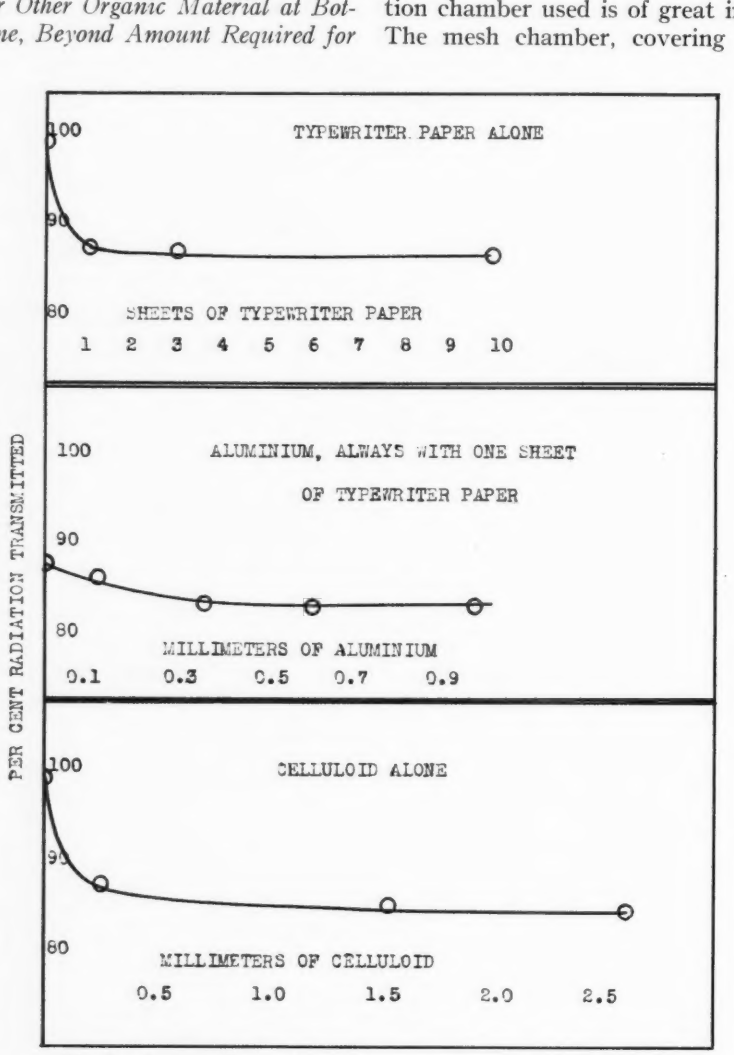

Fig. 2. Amount of secondary filter necessary at mouth of lead cylinder.

Secondary Filter.—As has just been shown, 1.5 mm. of bakelite or celluloid is a sufficient secondary filter at the mouth of a lead cone. However, considerably greater thicknesses are frequently used, sometimes more than a centimeter. This amount of organic material may, by scattering, produce definite changes in the beam of rays. Such changes would materially affect surface and depth doses in the tissues. For in- beam, registers the entire radiation which is to enter the surface of the body; inequalities in the distribution within the field are not indicated. On the other hand, the length of the path of the rays in this ionization chamber is important. If a ray, instead of going straight through the chamber traverses it diagonally, more ions are formed in the measuring volume, and a higher reading is the result; hence the

effect in the mesh chamber of increasing the thickness of organic material at the mouth of the cone is twofold. The amount of radiation reaching the chamber is decreased by absorption in the material. At the same time, by scattering, the paths of many rays are rendered oblique, thus increasing the ionization. These two effects may counteract each other, or either one may predominate, depending on the voltage, filtration, and geometrical conditions. Similar compensation will take place in irradiated tissues, but the values will not be the same as in the ionization chamber, because of the difference in density between air and tissue.

With the small "air-wall" chamber, the difference in length of path is not present. However, the reading in such a chamber will be affected by scattering in the thick secondary filter, partly because regions of the filter not directly above the chamber scatter rays into it, and partly because the scattered radiation, being softer, is more effective in producing ionization. Since most calibrations and depth dose measurements are made with small chambers of this type, and it is in these results that the presence of the thick secondary filter may introduce complications, the problem has been investigated with such a chamber.

With the 8.8 cm. lead cylinder, no top diaphragm in place, at 165 kv. and 0.54 mm. copper filter, measurements were made directly below the opening, and at a distance of 10 cm. below it, with the minimum secondary filter, and with a filter of 13 mm. of bakelite. Readings were also made on the surface and at a depth of 9 cm. in the paraffin phantom, for the two conditions. All the results are given in Table I.

The first half of the table shows the variation from the inverse square law caused by the scattering in the thick filter.

TABLE I.—EFFECT OF THICK SECONDARY FILTER AT MOUTH OF LEAD CYLINDER

(165 kv. (const. pot.); 0.54 mm. Cu filter; target-surface distance 65 C.; cylinder diameter 8.8 cm. Small air-wall spherical ionization chamber, volume 1.545 cc.)

Effect in Air—Distance Alone				
Secondary Filter mm. Bakelite	Ionization Reading (Arbitrary Units)		Per Cent Decrease with Increase in Distance	Per Cent Decrease Calculated by Inverse Square Law
	Chamber in Air at Mouth of Cylinder	Chamber in Air 10 cm. below Mouth of Cylinder		
0*	297	220	74	75
3	290	208	72	
7	281	192	68	
13	268	167	62	
Effect in Paraffin—Depth Dose				
	Chamber on Surface of Paraffin at Mouth of Cylinder	Chamber Buried in Paraffin at 9 cm. Depth	Per Cent Depth Dose	
0	422	177	42	
3	414	169	41	
7	402	158	39	
13	385	141	37	

* The thickness of the air-wall chamber was equivalent to the necessary secondary filter.

Without it, the inverse square law holds. With it, the percentage of radiation at the greater distance is less than that given by cone. The thin filter was thought too frail, and a heavy one substituted. A calibration was made with the ionization chamber

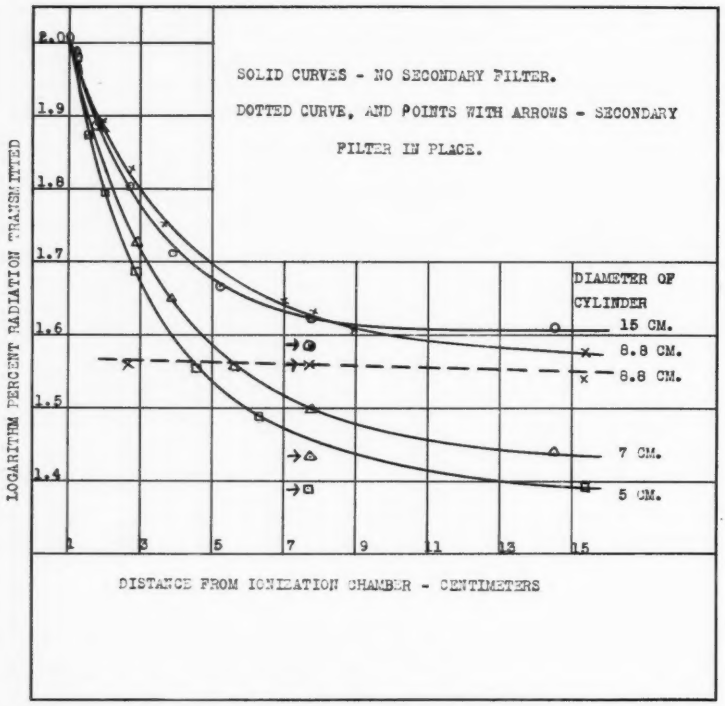

Fig. 3. Effect of distance from mouth of cylinder to ionization chamber.

the inverse square law. The greater the amount of bakelite present at the mouth of the cylinder, the greater the deviation from the inverse square law value. This means that the reading close to the filter is too high. In other words, the filter has scattered radiation into the chamber. The presence of the thick filter, while it ordinarily does not actually increase the radiation at the surface of the body, makes it relatively higher than readings taken at a distance from the mouth of the cone would indicate.

This phenomenon may readily cause errors in calibrations. An example of such an incident may be given. With a certain tube-holder having a thin secondary filter at the mouth of the attached cone, the inverse square law was found to hold for different distances below the mouth of the at a distance of 10 cm. from the mouth of the cone, the contact value then being calculated by the inverse square law. Treatments given on the basis of this calibration seemed to produce a more marked reaction than had been formerly obtained. Accordingly, a new calibration was made, this time with the chamber against the secondary filter. The resulting value was 15 per cent higher than the preceding one, which had been based on measurements at a distance from the cone. From the values in Table I, it can be seen that, with the set-up there employed, the use of 13 mm. of bakelite ($^1/_2$ inch) would have resulted in a discrepancy of 19 per cent. Three and 7 mm. (approximately $^1/_8$ and $^1/_4$ inch) would have produced discrepancies of 3 and 9 per cent, respectively. Evidently a thickness of about $^1/_8$ inch of bakelite to close the cylin-

der is not objectionable, but more than this should be avoided.

Returning to Table I, the second part presents the data regarding the effect of increasing the thickness of the organic secondary filter on the percentage depth dose. By increasing the effective surface dose, the filter causes a considerable reduction in the relative depth dose, in the extreme case for the experiments giving a drop of 13 per cent. The actual reduction will vary with the conditions of the irradiation, but the effect will always be present.

3. *Effect of Distance from Mouth of Cylinder to Mesh Ionization Chamber.*—As the ionization chamber is moved away from the mouth of the cylinder, two things happen. Any very soft radiation present suffers absorption by the increasing thickness of air. Also, due to change in the geometrical relations between the mesh and the opening of the cylinder, the amount of both scattered and secondary radiation reaching the chamber varies. If no secondary filter is used at the mouth of the cylinder, the readings drop rapidly, due mainly to the absorption of the electronic secondary radiation by the air. With the secondary filter eliminating this radiation, the change is almost entirely due to the shift in the relative positions of cylinder and chamber. The solid curves of Figure 3 show the drop in radiation with increasing distance from the lead cylinders, when no secondary filter is used. The dotted curve is for radiation from the 8.8 cm. cylinder when the secondary filter of 0.36 mm. Al plus 1 sheet of paper is in place. The extra points at 7.7 cm. distance, designated by arrows, are the values for the other cylinders at this distance when the secondary filter is inserted. It appears that for the smaller ones, 15 cm. of air and the altered geometrical relations remove the same radiation as the secondary filter, while a greater distance is necessary for the larger ones. Since the radiating source is not a point, but in addition to the target includes the primary filter, the secondary filter, and the cylinder walls, the geometrical effect of the change in relative positions is different for each size of cylinder.

4. *Position of Primary Filter.*—It is obvious that the secondary filter should be placed at the bottom of a lead cone. The proper position for the primary filter is not self-evident. It might be expected that in the secondary or scattered radiation from the cone, there would be components too hard to be taken care of by the aluminium and paper secondary filter discussed above, but so soft as to be undesirable in therapy. In this case, putting all or part of the copper at the lower end of the cone might be advantageous. On the other hand, radiation arising in the copper filter, or scattered by it out of the beam, might give undesirable results. In order to investigate this point, a copper filter made up of a number of thin sheets was used. With the secondary filter in place at the bottom of a lead cylinder, readings were made first with all the copper at the top, and then with gradually increasing amounts shifted from there to a position just above the aluminium. No top diaphragm was used, in order to have the extreme conditions of scatter from the cylinder walls. With the 8.8 cm. lead cylinder, it was found that a shift of 0.02 mm. copper from above to below made no difference in the reading. With a shift of 0.15 mm. copper, the reading in the mesh ionization chamber increased, indicating scattering in the copper, rather than absorption of any radiation given off from the lead. A similar result was obtained with the bakelite cylinder. When a limiting diaphragm is used at the top of the cylinder, so that little of the primary radiation strikes the walls, no difference in effect should be expected. However, the test was repeated for these two cylinders (lead and bakelite), with the appropriate limiting diaphragm, and the result was the same. According to this evidence, the primary filter should be placed at the top of any attachment of the type discussed in this paper.

5. *Quantity of Radiation Scattered by Collimating Devices.*—In order to investigate the actual contribution to the beam by the attachments, the tubes of the 8.8 cm. lead and bakelite cylinders were made

detachable. The secondary filter of 0.36 mm. aluminium and a sheet of paper was placed below, at a distance of 4.5 cm. from the mesh of the ionization chamber, and the usual filter of 0.54 mm. copper above, no limiting diaphragm being used. Readings were made with no collimating tube, and with the lead and the bakelite ones. The results are shown in Table II.

TABLE II.—AMOUNT OF RADIATION SCATTERED FROM CYLINDERS

165 kv. (Const. Pot.); 0.54 mm. Copper Filter; 8.8 cm. Cylinders		
Tube	Reading	Per Cent
None	470	100
Lead	480	102
Bakelite	495	105

Evidently the bakelite scatters somewhat more than the lead, which was to be expected, but in any case the contribution from the walls is very small when the tube is cylindrical. If the walls flare to fit the beam, it will probably be even less.[2]

No appreciable radiation, of course, passes through the walls of lead cones. A cone of bakelite, even with a suitable limiting diaphragm at the top, designed for use mainly as a pointer, will be penetrated by some rays. The total amount of this radiation transmitted by the walls can be determined by taking readings with and without the guard diaphragm at the bottom. For the 8.8 cm. bakelite cylinder, with the top diaphragm in place, the reading increased 17 per cent when the guard was removed. That is, the total radiation passing through the walls of this cylinder is 17 per cent of that in the desired beam. This 17 per cent was distributed over the entire area of the ionization chamber outside the direct beam. (See Fig. 1.) Any small portion of the skin just outside the beam would, therefore, receive only a very small amount of radiation. The test was not made without a limiting top diaphragm, since obviously such a set-up would never be used in practice.

6. *Effect of Collimating Device on Quantity of Radiation in Beam.*—Since the source of radiation is not even approximately a point, but includes regions off the focus of the target, and even on its back and stem, there is considerable difference in the amount of radiation delivered by the collimator if it is designed to fit the geometrical beam from a point source, or if it is simply a right cylinder with no limiting diaphragm at the top. The insertion of the appropriate limiting diaphragm affects the quantity considerably. Table III shows the effect of the diaphragms on the radiation

[2] The type of cone which tapers toward the bottom to reduce the area of the beam was not investigated. With such a cone, the proper diaphragm should be used at the top, to reduce the beam, so that the cone serves mainly as a pointer.

TABLE III.—EFFECT OF LIMITING DIAPHRAGMS ON AMOUNT OF RADIATION TRANSMITTED BY CYLINDERS

165 kv. (Const. Pot.); 0.54 mm. Copper Filter; Distance from Target to Top of Cylinder, 35 cm.							
Cylinder		Reading		Per Cent		Average per sq. cm.	
Diameter cm.	Area sq. cm.	With Diaph.	Without Diaph.	With Diaph.	Without Diaph.	With Diaph.	Without Diaph.
5	19.7	90	110	100	122	4.6	5.6
7	38.4	179	223	100	124	4.7	5.8
8.8	61	298	371	100	124	4.9	6.1
15	177	930	1140	100	123	5.2	6.4

from all the lead cylinders, the primary filter of 0.54 mm. copper being at the top, and the usual secondary filter of 0.36 mm. aluminium plus a sheet of paper at the bottom.

The increase in radiation on removing the diaphragm is practically the same in all cases, about 23 per cent, under the conditions of these experiments. The actual increase in any particular case is, of course, dependent on the distance from the top of the diaphragm to the target, on the type of x-ray tube, etc. In any case, there will be an appreciable amount.

From the point of view of quantity of radiation available, it is preferable to use no limiting diaphragm at the top of the cone. Also, the value *per square centimeter of irradiated area* shows a continual increase with the size of the cylinder. The rate of this increase is practically the same whether the diaphragm is used or not. In either case, it is to be attributed to the inclusion of more and more off-focus (stem, etc.) radiation in the beam.

The data on effect of limiting diaphragm on quantity of radiation in the beam were checked by means of a small spherical "air-wall" chamber placed at the center of the beam, just below the 7 and 15 cm. cylinders. For each, the increase in the reading when the limiting diaphragm was removed was found to be about 17 per cent. Since the small chamber measures radiation only at the center of the beam, and in the mesh chamber the effect at the edges is accentuated by the increase in path of the oblique rays, the two would not be expected to agree; the mesh chamber should (and does), give the higher value.

It may be noted that a certain increase in the intensity of the beam measured in free air (no attachments) occurs with increase in the area of the beam, when other parts of the target besides the focal spot are radiating. This has been shown by Thoraeus (3), and verified in the course of this check with the small chamber. With increase in the diaphragm (at 35 cm. from the target) from 7 to 225 sq. cm., no cone being used, the intensity at the 65 cm. distance, in the center of the beam, increased by 19 per cent. It is evident from this that calibration of the tube output, even in air, should be done for different beams. If there is a diaphragm very near to the x-ray tube, so that off-focus radiation is practically eliminated, this difficulty does not arise.

7. *Quality of Radiation Transmitted by Collimating Devices.*—If the off-focus radiation, which may be a significant part of the beam, as shown in Section 6, were of very different quality from that coming from the focal spot, its admixture would be undesirable. It has been shown by Taylor (4) that radiation from different parts of the target differs somewhat in quality. The difference is not great enough to be considered with regard to a possible differential effect of various qualities of radiation; its only significance would be whether it might have an effect on the relative depth dose. Accordingly, this was investigated, with the small ionization chamber and paraffin phantom, for the 15 and 7 cm. cylinders, depth doses being measured with and without the limiting diaphragms in place. The results are given in Table IV.

TABLE IV.—EFFECT OF THE PRESENCE OF OFF-FOCUS RADIATION ON DEPTH DOSE

165 kv. (Const. Pot.); 0.54 mm. Copper Filter; 65 cm. T-S Dist.				
Position of Chamber	15 cm. Cylinder		7 cm. Cylinder	
	With Diaph.	Without Diaph.	With Diaph.	Without Diaph.
Surface	4.85	5.48	3.85	4.32
9 cm. Depth	2.52	2.78	1.38	1.53
Per Cent Depth Dose	52	51	28	28

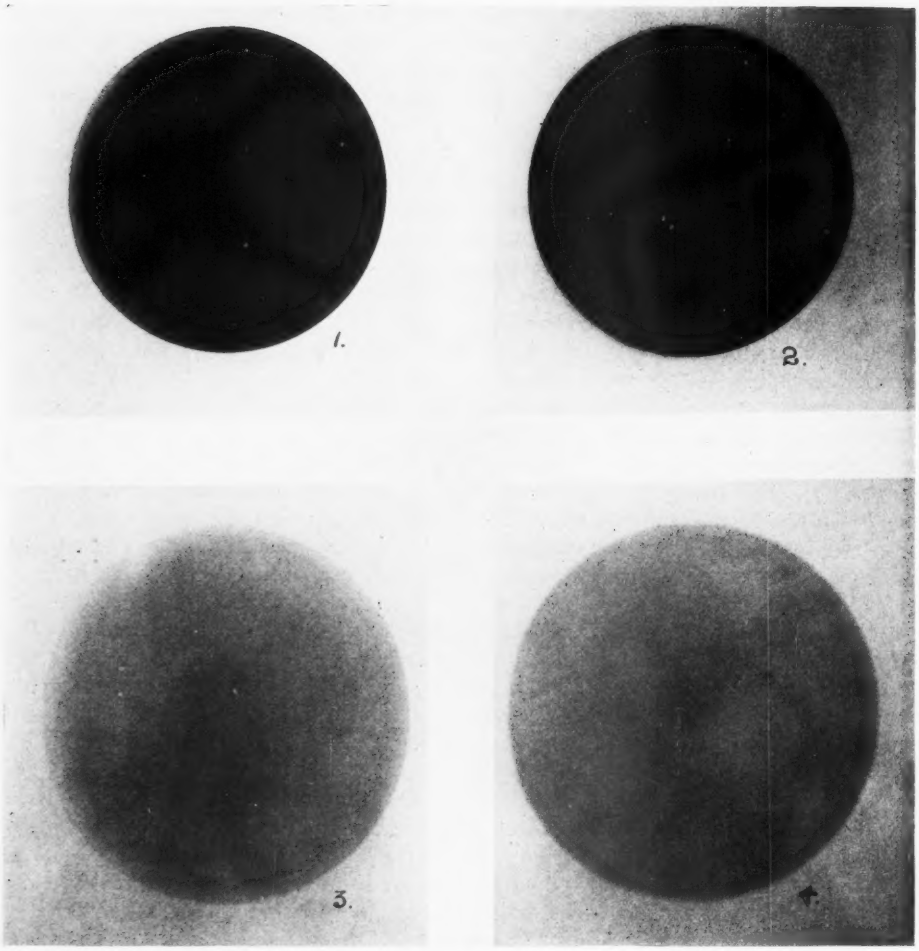

Fig. 4. Beam from 8.8 cm. lead cylinder in paraffin phantom: 165 kv., 2 ma., 0.54 mm. Cu at top of cylinder: 0.36 mm. Al plus 1 sheet paper at bottom; 65 cm. target-phantom distance.

Position of film:

1. On surface of paraffin; limiting diaphragm at top of cone.
2. On surface; no diaphragm.
3. At 9 cm. depth in paraffin; diaphragm in place.
4. At 9 cm. depth; no diaphragm.

The readings are given in arbitrary units, and the percentage depth doses calculated for the two cases. It is evident that, judged by this criterion, the admixture of off-focus radiation produced by removing the limiting diaphragm does not materially alter the quality of the beam. The depth dose is practically the same, whether the diaphragm is used or not. Since the quantity of radiation available is greater when the diaphragm is omitted, it would seem more advantageous from the point of view of efficiency not to use it.

With regard to the effect of the cone itself on the quality of the radiation, no direct measurements have been made. As has been shown above, when copper is moved from the top to the bottom of the cylinder, the reading is increased, due to scatter from the filter. Hence a comparison of absorption data obtained with the copper in the two positions would have no

meaning with regard to the relative qualities of the beam with and without the inclusion of any radiation from the cylinder walls. It is, however, possible, by taking such pairs of curves for the lead and bakelite cylinders of the same size, to see whether the effects produced by the two are different. It is hardly to be expected that they would be, for the scattered radiation from their walls has been shown above to play a very insignificant rôle. However, for these two cylinders, and for both 165 and 200 kv., absorption curves were made, with no top diaphragm, and with the copper first above and then below. In all cases, for 165 kv. every point on a curve for the bakelite cylinder was from 2 to 3 per cent higher than the corresponding point with the lead cylinder; for 200 kv. the difference was from 3 to 4 per cent. These differences represent the higher scatter from the bakelite cylinder. Their constancy regardless of the thickness of the copper absorber indicates that there is no appreciable difference in the quality of the total radiation passing through the lead and the bakelite tubes.

8. *Spreading of the Beam within the Tissues.*—One object of limiting the beam is to restrict the radiation as much as possible to the diseased region and to keep it from normal tissues. If the undiaphragmed cylinder delivered a markedly higher dose of radiation to tissues outside the direct beam, it would be a disadvantage. To investigate this point, four films were exposed for exactly the same times, under the following conditions:

1. On surface of paraffin; limiting diaphragm in place at top of cylinder.
2. On surface; no diaphragm at top of cylinder.
3. At 9 cm. depth in paraffin; diaphragm in place.
4. At 9 cm. depth; no diaphragm.

The results are shown in Figure 4. In the photograph it is difficult to see any difference in Nos. 3 and 4, so far as the radiation outside the direct beam is concerned. In the original films, No. 4 is slightly darker throughout, but the difference is very small. It was not considered necessary to attempt to photometer these films and obtain a numerical comparison, since the effect is evidently slight.

PRACTICAL APPLICABILITY OF PRECEDING DATA

The experiments herein discussed were performed with the special set-up described at the beginning of the paper, which is quite different from many therapeutic installations, particularly with modern shockproof tube-holders. The applicability of these results to such installations may be questioned. However, it may be stated that much of the information here given holds good for any type of tube-holder and cone in use. Some differences may arise, when there is a diaphragm closer to the x-ray tube than has been used in this work, so that most of the off-focus radiation is excluded. In that case, the data obtained for the cylinders without limiting top diaphragms have no significance. The information obtained with the limiting diaphragms in the tops of the cylinders is applicable to any cone set-up in which the beam is effectively limited to the focal radiation. This includes the amount of secondary filter necessary, the harmful effect of too much secondary filter, the position of the primary filter, and the transmission of radiation by the walls of light cones used as localizers, as well as the effect of the cone walls on quality and quantity of transmitted radiation.

SUMMARY AND CONCLUSIONS

Certain practical problems have been investigated regarding the use of devices to localize or collimate the beam of radiation in x-ray therapy (ordinarily spoken of as "cones," regardless of their actual shape).

The filter required at the bottom of lead cones, to remove soft secondary radiation, is 0.3 mm. aluminium plus a sheet of paper, or thin layer of celluloid. If celluloid or bakelite alone is used, a thickness of about 1.5 mm. is necessary and sufficient. For bakelite cones, used only as localizers, no

secondary filter is required, provided they are at least 15 cm. long.

The use of a thick layer of bakelite or other organic material at the mouth of a cone decreases the relative depth dose. The use of such a thick secondary filter is discouraged. If, however, one is employed, care should be taken that, in calibrating, the ionization chamber is placed very close to it.

The primary filter should always be at the top of the cone, to avoid scattering from the copper.

When cylindrical attachments are used, with no limiting top diaphragm, the scattering from their walls is negligible, being about 2 per cent for lead and 4 per cent for bakelite. If the cone is flared to fit the geometrical beam, or a limiting diaphragm is used, the scatter is even less.

The amount of radiation passing through the walls of a bakelite cone (limiting diaphragm at top) is small in comparison to the direct beam (17 per cent for the particular case, which is probably as much as will be obtained in any therapeutic setup). The use of cones of light material, as pointers, is satisfactory if the proper limiting diaphragm is used near the x-ray tube.

With lead cones of sufficient wall thickness, the use of a limiting diaphragm at the top to define the x-ray beam is unnecessary. It reduces the radiation considerably, by cutting off rays from the back and stem of the target. The relative depth dose is not improved by eliminating this radiation.

The material of the cone itself has no appreciable effect on the quality of the total radiation passing through it.

The spread of the beam within the tissues is not notably greater when the limiting diaphragm is omitted.

The authors wish to acknowledge their indebtedness to Dr. Failla, who suggested the problem, for his assistance and advice throughout the course of the work.

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RIGHT AORTIC ARCH

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RIGHT aortic arch is a rare anomaly, the diagnosis of which is seldom established during life. Paradoxically, it is the easiest of all the congenital anomalies of the circulatory system to recognize with certainty. This can be accomplished immediately in practically all cases in which a complete roentgenologic examination of the heart can be made. The importance of this becomes more manifest when we realize that the diagnosis cannot be established with any reliance by the usual clinical procedures. All cases familiar to the writer, which have been identified prior to the findings of the autopsy table, were diagnosed through the use of the roentgen ray. However, there is a strong likelihood that many cases of right aortic arch have passed through the hands of the roentgenologist without having been recognized. The factors responsible for this condition are not hard to find: (1) Elimination of fluoroscopy from the examination; (2) restriction of the radiographic examination to the postero-anterior film; (3) failure to consider barium studies of the esophagus as a part of the cardiac examination—particularly in obscure cases.

The object of the present paper is not so much to add another case to the literature of this rare anomaly as to forward a plea for a complete examination of all cardiac cases. The writer is of the firm opinion that not less than two exposures are necessary for even a cursory examination of the heart. The ones of greatest value can never be determined in advance, the selection requiring a preliminary fluoroscopy. The fluoroscopy employing the different oblique positions and including barium studies of the esophagus should be carried out in every complete cardiac examination in which an abnormality of the arch or one of the great vessels is suspected.

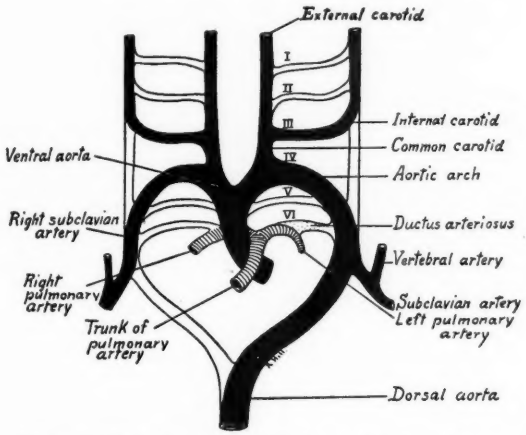
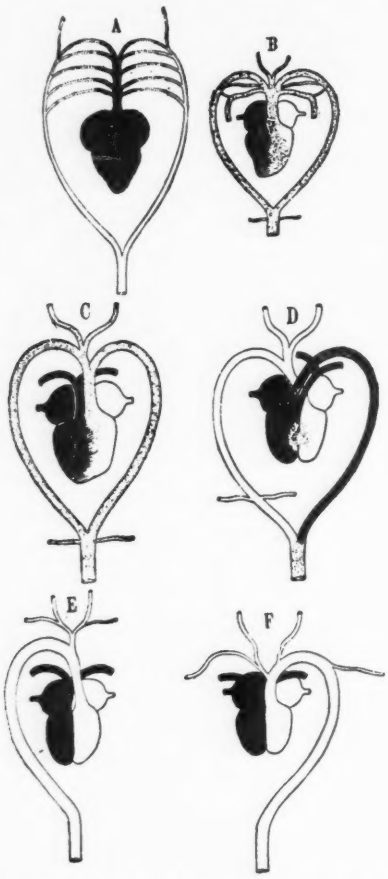


Diagram showing the aortic arches and their derivatives in human embryos.

Fig. 1. (*See Footnote 1.*)

Excellent reviews of the embryology of this anomaly are found in other articles, and no attempt to redescribe these aspects will be made except to remind the reader that of the six paired aortic arches of the embryo, the fourth left aortic arch becomes

the definitive arch of the normal adult. Exceptionally in man and constantly in birds the fourth right aortic arch becomes the



Diagrams of the heart and its compartments in the different vertebrate Classes. [In part after WIEDERSHEIM.]
(A) Fish; (B) Urodele; (C) Anuran; (D) Reptile; (E) Bird; (F) Mammal.
Fig. 2. (*See Footnote 1.*)

functioning unit, the left arch undergoing atrophy either in whole or part (Figs. 1 and 2).[1]

Blackford and others, in 1932, write that at least a hundred cases of this anomaly are on record. The few identified during life have been reported almost exclusively in foreign periodicals. Notable exceptions to these are the cases of Blackford (3), Jenkinson (7), and Sprague, Ernlund, and Albright (11). The present case was recognized during the course of a routine gastrointestinal examination, the patient having no cardiac complaints.

Clinical History.—The patient was a woman of 45 years, who entered the hospital with a history of gradual failure of health since hospitalization for sepsis (right arm) nine years before. She complained chiefly of headache and abdominal pain. Her past and family histories were not significant. Few positive findings were noted on physical examination. The heart was

[1] Figures 1 and 2 were taken from Prentiss' Embryology and Wilder's History of the Human Body.

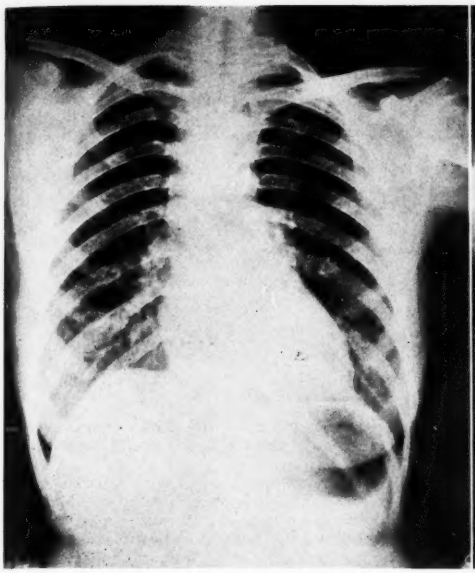


Fig. 3. Postero-anterior film. Positive findings in this projection are: (1) absence of the aortic knob; (2) prominent vascular shadow to the right of the spine, fading out beneath the medial end of the right clavicle; (3) displacement of the esophagus (and trachea) to the left; (4) temporary obstruction in the esophagus at the level of the arch, indicated by barium shadows.

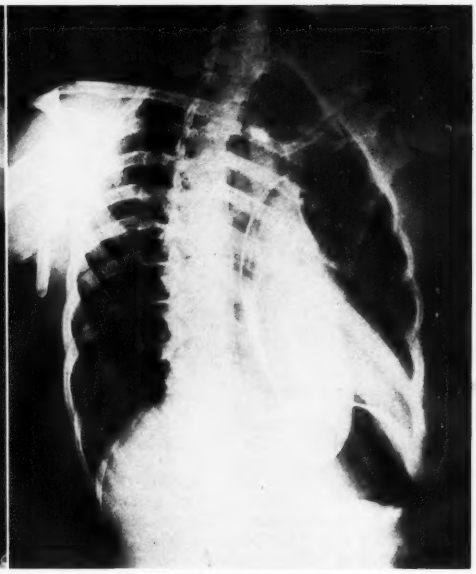


Fig. 4. Right anterior oblique film. (1) Hemispherical pressure defect at the level of the aortic arch; (2) temporary obstruction to the passage of barium at this level, and (3) clouding of the mediastinum, due to the anomalous position of the descending aorta.

not enlarged. A systolic murmur was heard over the cardiac area inside the nipple line and over the pulmonic area. There was a slight roughening of the first sound over the aortic area which was not transmitted toward the vessels of the neck. The blood pressure was found to be 140 systolic and 86 diastolic.

Roentgenological Examinations.—A gastro-intestinal series was requested mainly because of the history of a loss in weight and abdominal pain. The stomach appeared normal in contour, without any evidence of filling defects. Ptosis was present. Motility studies were normal. The only unusual feature related to the esophagus at the level of the aortic arch (Figs. 3 and 4). Beside a temporary delay at this level, a smooth hemispherical defect was apparent on the posterior wall of the esophagus, suggesting external pressure by an adjacent tubular structure. It was this finding which led to a complete chest examination, including left and right anterior oblique films.

Chest.—The postero-anterior film showed a heart which appeared slightly increased in size, and presented a shape consistent with an early left ventricular hypertrophy. Considering the patient's age, a more striking finding was the absence of the aortic knob (the shadow of the descending limb of the aortic arch). A prominent vascular shadow to the right of the spine at the level of the great vessels ran upward toward the medial end of the clavicle, fading out at this level (Fig. 3). During the fluoroscopy, a rather vigorous systolic pulsation was evident in this region. The trachea was noted to be slightly displaced to the left at the clavicular level. The left margin of the great vessels was very straight from the level of the arch down to the cardiac shadow. The pulmonary artery was inconspicuous. Erosions of ribs were not noted. The right anterior oblique film

Fig. 5. Fig. 6.

Fig. 5. Left anterior oblique film, showing the failure of the descending limb of the aortic arch to outline in its usual position (over spine). Note that the plane of displacement of the trachea is such that it cannot be identified in this projection.

Fig. 6. Left oblique film, showing the upper portion of the esophagus partly filled with barium. Note the absence of any pressure defect on the posterior aspect of the esophagus above the arch by an anomalous branch of the aortic arch. This suggests that the left subclavian in this case either arises from an aortic diverticulum at the semicircular defect or that the case is one of symmetrical reversal. Since there is a large pressure defect of the esophagus at the level of the arch and the descending aorta does not appear to pass behind the esophagus but rather descends to the right of the spine, the former of these two possibilities appears more likely.

(Fig. 4) showed anterior displacement of the trachea at the level of the arch. The mediastinum in this position did not present its usual clear appearance; it was obscured down to the level of the heart by a homogeneous shadow, the density of which was similar to that of the vascular shadow. This appeared to be due to an anomalous position of the aorta; it ended abruptly superiorly at the level of the arch. The left oblique film (Figs. 5 and 6) was chiefly important in demonstrating the failure of the descending limb of the aorta to outline in the usual position. This limb of the arch usually overlaps the spine slightly, and in normal cases is outlined without difficulty if satisfactory films can be obtained. Likewise, the usual clear-cut outline of the right innominate vessel proceeding toward the head and neck was absent. The shape of the heart confirmed the impression obtained from the postero-anterior film that the left ventricle was somewhat hypertrophied.

Esophageal Studies.—During the ingestion of barium, the esophagus was observed to be displaced toward the left side of the body at the level of the arch, the displacement amounting to approximately 1.5 cm. (postero-anterior film). Temporary delay in the passage of barium was apparent. The main bulk of the barium slipped down slowly, leaving a small residual amount suspended at the level of the arch, which would often remain in this position for several minutes. The swallowing of more barium in the erect position resulted in sufficient intra-esophageal hydrostatic pressure to quickly force the main bulk of the material into the lower esophagus, again leaving a small residue at the level of the arch. The actual mechanical relationships were best studied with the patient in the right anterior oblique position. The esophagus descended behind the trachea down to the arch. At this level, it acquired a more lateral position so that, when studied in the right anterior oblique position, the shadow of the esophagus was superimposed upon that of the trachea. This was produced by an abrupt change in the course of the esophagus at the level of the arch, which resulted in a smooth sharply defined curvilinear defect of hemispherical contour being produced upon the right lateral and posterior walls of the esophagus. At the

TABLE I.—ANATOMICAL RELATIONS BETWEEN THE SUBCLAVIAN ARTERIES AND THE ESOPHAGUS

Type of Origin	Side and Place of Origin	Transposition to Opposite Side	Order of Origin of Great Vessel	Deformity of Esophagus or Trachea	Position of Aorta
Right Subclavian					
(1) Branch of double aortic arch	Right of mid-line lateral to trachea and esophagus	Absent	One of the two vessels of the posterior or right arch	None by rt. subclavian; pressure defect on posterior wall of esophagus by posterior arch	The two arches surround the esophagus and trachea by an arterial collar
(2) Persistent rt. aortic root with normal left arch	Left of mid-line in distal portion of arch	Present	Last	Posterior pressure defect of esophagus	Relation of aorta normal
(3) Separate vessel of rt. aortic arch	Right	Absent	Next to last	None by rt. subclavian (pressure defect, however, by left subclavian passing behind esophagus)	Aorta descends looping to the right of spine
(4) Symmetrical reversal	Right	Absent	Last	None	Descending aorta to right of mid-line
(5) Branch of rt. innominate	Right (rare)	Absent	First	None by rt. subclavian	
Left Subclavian					
(1) Branch of double aortic arch	Close to mid-line in front of trachea	Passes to left	Both left carotid and subclavian arise close together near mid-line from left aortic arch	Left subclavian not in contact with esophagus. Pressure defect on esophagus by posterior arch, and possibly on trachea by anterior arch	The two arches surround the esophagus and trachea, the smaller arch passing anteriorly
(2) Separate vessel of left aortic root	Close to mid-line behind esophagus	Passes to left	Last	Pressure by aortic diverticulum on posterior esophagus	Descending limb of arch passes to right
(3) Incomplete double arch with patent ductus	Rt. of mid-line left subclavian arises from persisting anterior portion of left aortic arch	Present	Last	None by subclavian, but posterior aspect of esophagus may show impression of aortic arch	Aorta commonly descends to left of mid-line; right arch passes behind esophagus, left arch persisting to ductus forming subclavian
(4) Branch of left innominate: symmetrical reversal	Close to mid-line	Passes to left	First	Left subclavian passes in front of trachea. No deformity of esophagus	Descending limb of arch passes to right
(5) Separate branch of rt. aortic arch	Right	Present	Last	Pressure defect possible on posterior wall of esophagus by left subclavian	Descending limb of arch passes to right

level of the defect, the barium passed through the esophagus as a very thin stream, suggesting marked compression with forced collapse of the walls of the tube by external pressure. Behind and slightly lateral to the esophagus was the shadow suggesting an anomalous portion of the aortic arch which was responsible for the clouding of the upper part of the mediastinum. Below the level of the aortic arch, the esophagus gradually acquired its normal relationships within the posterior mediastinum. These profound changes in the shape and course of the esophagus may be easily missed in the left oblique position, the plane of displacement being the same as that of the beam of x-rays.

Discussion of Roentgenologic Findings.—The findings may be conveniently subdivided into those due to (*a*) the anomalous course of the right aortic arch itself; (*b*) the coarctation, a concomittant feature of right-sided aorta; (*c*) the anomalous origin of the main branches of the aortic arch.

SIGNS RELATING TO ANOMALOUS COURSE OF THE AORTA

(*1*) *A Prominent Vascular Shadow to the Right of the Spine at the Level of the Great Vessels* (*Postero-anterior Film*).—This shadow with its systolic pulsation is due to the aberrant course of the ascending aorta, as a result of the arch passing or hooking over the right main bronchus. The upper component of this shadow (the portion extending above the level of the arch) is probably cast by one of the great arterial branches of the arch.

(*2*) *Displacement of the Trachea and Esophagus to the Left* (*Postero-anterior Film*).—The hooking of the aorta over the right main bronchus results in sufficient mechanical pressure to displace the structures of the superior mediastinum of which the trachea and esophagus serve as roentgenologic landmarks. Of these two structures, only the displacement of the trachea can be identified, of course, in the usual radiographs, though the extent of the displacement is commonly greater in the case of the esophagus (if barium has been administered to permit the outlining of the esophagus). The esophagus is a more flexible or mobile structure, and the effects of mechanical pressure produce a greater displacement and deformity of contour in the organ. The changes in the trachea might in some cases escape attention unless particular care is exercised, but the changes in the esophagus are certain to be recognized if one carries out a complete examination with barium.

(*3*) *Displacement of the Esophagus Anteriorly* (*Right Anterior Oblique Film*).—This may be due to pressure of the right aortic arch, or a persisting remnant or diverticulum of the left aortic arch. However, this should not be considered as the sole cause in all cases. Other possibilities are a left subclavian artery or a patent ductus. The size and shape of the defect in the present case suggests that the defect is due to the aorta—either the right arch or a diverticulum of the left arch.

(*4*) *Hemispherical Defect of Oblique Films.*—The defect shows smooth sharply defined margins. The sweep of the arc and its location at the level of the arch gives one the impression that it has been produced by a tubular structure. The size of the shadow, particularly the magnitude of the arc, definitely suggests a defect resulting from aortic pressure. A large arc is indicative of either a large right aortic arch or a diverticulum of the left aortic arch. A small arc suggests that there is either a double arch with the resulting splitting of the arterial stream, or that the defect has been produced by a smaller vessel than the aorta, *i.e.*, the left subclavian or patent ductus. The latter is a very rare possibility and thus far has not been observed in man. The general size of the defect should not be confused with the magnitude of the arc. By the latter is meant the radius of curvature of the arc.

Signs Relating to Coarctation of the Left Aortic Arch.—These signs have been discussed in a previous communication (5), and require only a brief comment for present purposes. These are: (1) absence of the aortic knob (postero-anterior film); (2) failure of the descending limb of the aortic

arch to outline (left anterior oblique film); (3) absence of the usual aortic impression on the anterior wall of the esophagus as observed in the right oblique position. It should be noted that these signs are all based on negative findings, but it is the writer's conviction that a positive diagnosis of coarctation based on all three of these negative criteria may be made with great accuracy if satisfactory films can be obtained. Caution is urged in interpreting individual signs. It should be remembered that the absence of the aortic knob (posteroanterior film) is a constant normal finding during youth. Films of high technical quality are required for visualization of the arch in the left oblique projection. In properly exposed films, the descending limb of the aortic arch (convex border) is observed to overlap the upper dorsal spine. In right-sided aorta this part of the spine appears entirely clear. The descending portion of the aorta, if visualized at all, will be observed far to the right, entirely free and clear of the spine. If identified, this shadow represents the descending limb of a right aortic arch. Blackford and his coworkers (3) identified this shadow on their films, and were able to follow its continuation with the descending aorta downward through the overlapping shadow of the heart. Such a shadow, if it can be positively identified as the aorta, is convincing proof of a right-sided aorta.

ANOMALOUS ORIGIN OF GREAT VESSELS OF THE AORTIC ARCH

Portions of the outlines of the great vessels may be identified on both the posteroanterior and oblique films, but it must be admitted at once that seldom can we ever hope to hazard an opinion concerning their identity by direct inspection of their roentgenologic shadows. However, since the anomalous origin of these vessels may lead to pressure defects on the esophagus, the roentgenologist should be familiar with the common anomalous variations, and it is not inconceivable that a filling defect of the esophagus, combined with a knowledge of their probabilities, will enable one to ad-

TABLE II.—RELATIONS BETWEEN THE CHARACTER OF THE DEFECTS AND THE TYPE OF ANOMALY

Abnormal Pressure Defect of Esophagus at Level of Arch	Pressure Defect of Esophagus Above Arch	Size of Defect	Anterior Displacement of Esophagus	Course of Descending Aorta	Diagnosis
Present	None	Large, deep, wide arc	Present	Anomalous to rt. of normal position	Right aortic arch, with persistent left aortic diverticulum
Present	None	Large compatible with aorta	Present	Passes to left side behind esophagus	Right aortic arch, with persistence of ventral portion of left arch
Present	None	Smaller than defect expected in normal aorta	Variable	Passes downward to left of mid-line	Double aortic arch
Absent	On posterior wall, less often anterior wall of esophagus	Small, shallow by right subclavian	Absent	Normal	Persistent right aortic root
Present	On posterior wall of esophagus	Small, shallow by left subclavian, wide arc by aorta	Absent	Anomalous	Right aortic arch, with separate origin of main branches from rt. aortic arch
Present	None	Wide arc by aorta	Absent	Anomalous	Right aortic arch, with symmetrical reversal of vessels

vance a shrewd guess that an anomalous variation of the arterial branches is present, limited to several likely types of such variations.

It is convenient for purposes of this discussion to describe these anomalies with special reference to the subclavians. The left subclavian in cases of right aortic arch may arise as follows:

(*a*) *A Separate Vessel of the Left Arch in Cases of Double Aortic Arch.*—The persisting double aortic arches form an arterial collar about the esophagus and trachea. The main left branches arise from the arch in front of the trachea and do not come in contact with the esophagus. The esophagus is pressed upon by the posterior or right arch. A semicircular pressure defect on the posterior wall of the esophagus, if noted in conjunction with a similar defect on the anterior wall of the trachea, points to the presence of a double arch. In most instances each of the great arterial branches courses independently from the respective arches without the formation of either a right or left innominate. Less commonly, as in the case of Biedermann (2), a double arch (with ductus arteriosis) may be incomplete with persistence of the left arch proximal to the origin of the ductus, the left subclavian passing from right to left in front of the trachea and the right subclavian arising from the right innominate (right arch). In this case the main arterial branches do not come in contact with the esophagus, but the aortic arch passes behind the esophagus. Since the entire output of the left ventricle is carried by this arch, the semicircular pressure defect should have a radius of curvature compatible with the size of the normal aorta. The only arterial branch in a position to produce compression is the left subclavian which might modify the anterior aspect of the trachea.

(*b*) *Separate Vessel of a Persistent Right Arch.*—The left subclavian, which is the last of the four main branches to be given off from the arch (in order, left common carotid, right common carotid, right subclavian, left subclavian), passes from right to left behind the esophagus. In this case the aorta descends to the right of the esophagus (not crossing to the left of the spine as in the cases above). The left subclavian being the last branch of the arch, has acquired a posterior position and passes behind the esophagus. Such a pressure defect, if identified, should be at the level of the arch or slightly above. A patent ductus may be present in this form of anomaly.

(*c*) *Separate Vessel Arising from Diverticulum* (the latter representing a persisting remnant of the left aortic arch).—The diverticulum is usually located behind the esophagus, and is one of the structures which may be responsible for the semicircular defect observed along the posterior wall of the esophagus at the level of the arch. The left subclavian, which finds its origin from the diverticulum in these cases, passes posteriorly to reach the left side of the body.

(*d*) *Branch of Left Innominate.*—In this form of variation, the left innominate is the first branch of the right arch, with the right carotid and right subclavian following in order (symmetrical reversal). The left subclavian, as a division of the left innominate, arises close to the anterior aspect of the trachea, and proceeds upward and laterally to reach the left side of the body. This type of anomaly is most apt to be complicated by other cardiovascular anomalies of a serious character.

At least four different types of origin are found when the right subclavian is considered.

(*a*) *Separate Vessel in Cases of Double Aortic Arch.*—The vessel arises to the right of the mid-line close to the right side of the trachea and proceeds upward and laterally without crossing the mid-line.

(*b*) *Separate Vessel of the Right Aortic Arch.*—Usually in this anomaly the right subclavian is the next to the last branch to be given off from the arch, except in cases of symmetrical reversal. Its point of origin in respect to the arch, however, lies to the right of the mid-line. It is usually the last branch, the left subclavian, which must cross the mid-line to reach the left side of

the body (in front of the trachea). In cases of symmetrical reversal, the right subclavian is the last branch, the arch which loops over the right bronchus to descend far to the right of its usual position. The right subclavian, therefore, does not cross either the esophagus or trachea.

(*c*) *Branch of Right Innominate.*—A right innominate artery usually is not formed in cases of right aortic arch, but in those rare cases in which it may occur there would be no occasion to expect that it would deform either the trachea or esophagus.

(*d*) *Persistent Aortic Root of a Right Aortic Arch.*—In this type of case, the aortic arch assumes its normal course, passing to the left in front of the trachea and hooking over the left bronchus. The right subclavian, however, is the last rather than the first branch of the arch (as a division of the innominate), arising at the junction of the transverse portion and descending limb of the arch, to the left of the mid-line. To gain the right side of the body it passes obliquely behind the esophagus at the level of the second to fourth thoracic vertebræ. Less commonly, it may pass between the esophagus and trachea, but rarely is noted coursing in front of the trachea. This vessel is in an excellent position to produce a pressure defect on the esophagus.

Relations between the Type of Anomalous Origin of Vessels and Pressure Deformities of the Esophagus and Trachea.—A separate origin of all four main branches of the aorta is the most common variation of the single right arch. An innominate, when formed, is usually the left; the formation of a right innominate in cases of right aortic arch is distinctly rare. When all four vessels of the right aortic arch are formed separately, the aorta loops over the right bronchus, passing to the right of the mid-sagittal plane of the body. If the aorta does not cross the esophagus at the mid-line, the left subclavian must cross the mid-line to reach its proper side, which is commonly accomplished by passing behind the esophagus (obliquely above the level of the aortic arch). When an innominate is formed, as in cases of symmetrical reversal, the left subclavian which arises from it (close to the mid-line) must cross the mid-line, but in this instance it passes in front of the trachea. In this type of case neither the aorta nor its main branches cross the mid-line in the region of the esophagus, and therefore no pressure defect on this hollow viscus need be expected, as in cases of separate origin.

In cases of complete double aortic arch, the main branches arise separately from their respective arches. The right aortic arch passes behind the esophagus, and, since it is commonly the larger of the two arches, it may produce a considerable defect on the collapsible posterior wall of this structure. Not uncommonly, the only part of the right arch which persists is the aortic root, the routing of the blood being normal through the left aortic arch. In this event the right subclavian arises on the left side of the mid-line as the last vessel and passes behind the esophagus to gain the opposite side. As a rare form of the incomplete type of double arch, the left arch may persist to the point of the ductus arteriosis (the rest of the arch disappearing) to give rise to the left subclavian. The left subclavian may arise on the right of the mid-line, but in crossing the mid-sagittal plane it passes in front of the trachea in this type of anomaly. Since the right arch is the functional arch, it lies posteriorly and crosses behind the esophagus to reach the opposite side. The left subclavian may arise from a persisting root of the left aortic arch in a manner just detailed for the origin of the right subclavian from a persisting root of the right aortic arch. The left subclavian arises close to the mid-line, often from a diverticulum behind the esophagus (representing the root remnant) and passes to the correct side of the body. Apparently the diverticulum rather than the left subclavian is responsible for the defect observed along the posterior wall of the esophagus.

In summarizing these changes, we may add that the right subclavian arises on the correct side of the mid-sagittal plane of the body in all cases of the right or double aortic arch, except in cases of a right persistent aortic root, and that the left sub-

clavian arises either close to the mid-line or on the contralateral side in all cases of either right or double aortic arches. To gain the correct side, the left subclavian passes in front of the trachea in cases of complete double aortic arch, and in cases of right aortic arch with symmetrical reversal (formation of left innominate). In other forms of right aortic arch, the left subclavian, as the last branch of the aortic arch, passes posteriorly behind the esophagus. In cases of incomplete double aortic arches, the left subclavian may pass either in front of the trachea or behind the esophagus, depending on the portion of the left arch which has undergone atrophy. For example, in cases of left persistent aortic root (with disappearance of the dorsal portion of the embryonic arch to the point of origin of the ductus arteriosis), the left subclavian has a deep position within the thorax and arises from a diverticulum behind the esophagus. In cases in which the reverse conditions obtain, the left subclavian arises anteriorly close to or slightly to the right of the mid-line and passes to the left in front of the trachea.

With this information at hand, many valuable deductions can probably be made regarding the type of pressure defect produced upon the esophagus (and, rarely, the trachea). If the defect is at the level of the aortic arch and presents a wide radius of curvature on the posterior aspect of the esophagus, it is apparent that the vessel is either a right aortic arch with a persistent left aortic diverticulum (a form of incomplete double aortic arches), a right aortic arch with persistence of ventral portion of the left arch, or a complete double arch. If the defect is smaller and at a higher level than the arch, the vessel responsible is one of the subclavians. It is the right subclavian if one can demonstrate an aortic arch with a normal course (aortic knob on the left, normal spatial relations in the oblique positions). If an anomalous course of the aortic arch is demonstrated, the defect is due to the left subclavian. In this event one may safely hazard the opinion that there is a separate origin for all four main branches of the aortic arch, the left subclavian being the last. If there is no pressure defect on the esophagus above the level of the aortic arch, and an anomalous course of the aorta can be demonstrated without any anterior displacement of the esophagus, the case is probably one of symmetrical reversal, with the formation of a left innominate.

Since the present case shows a broad defect at the level of the arch and an absence of any pressure defect in the esophagus by one of the subclavians (Fig. 6), the anomaly presumably is one of right aortic arch with persistent left aortic root, the latter serving as the origin for the left subclavian.[2]

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[2] This case was referred to us by Dr. P. F. Metildi, to whom we are indebted for the opportunity to make a detailed study of the heart. We are particularly thankful for the assistance extended by Dr. M. J. Gerbasi in helping us follow this patient after the preliminary studies were made.

THE RADIUM TREATMENT OF POST-OPERATIVE PAROTITIS[1]

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ALTHOUGH fortunately a rare complication, acute parotitis has for many years been recognized as a very serious post-operative infection. Because of the well-known association of epidemic parotitis with orchitis or oophoritis, operation on the genital organs was considered the type of surgery most likely to be followed by acute infection of the parotid glands. In 1878 and 1880, German authors reported cases following ovariotomy (2). Blair and Padgett discussed the history and etiology of the condition and gave their results; 14 cases of post-operative parotitis were cited, with seven deaths, a mortality of 50 per cent. Rankin and Palmer found 79 cases in a review of the literature, with 36 deaths, or a mortality of 46 per cent. Green, in 1935, cited the mortality in the combined American statistics as 58 per cent, individual reports varying from 25 to 60 per cent.

The incidence of the complication is low, since it was found to occur following about one in 2,275 general operations (1). Rankin and Palmer, however, found operations on the colon and rectum rather than pelvic operations to be the surgical procedures more often followed by acute parotitis. The complication was seventeen times as frequent after operations on the colon as after general surgical procedures.

Regarding etiology, five theories have been advanced. Only two of these are important: (1) the pyemic theory, according to which a septic clot from the primary focus of infection forms an embolus in a parotid vessel, and (2) the theory of infection of the duct, according to which secondary parotitis is caused by direct extension of micro-organisms along Stensen's duct from the mouth. The latter theory is supported by the experimental work of Claisse and Dupré (3 and 4), in 1894, who found they could infect the glands of dogs with micro-organisms only injected into or spread over the duct while the animals were either starved or while their secretions were diminished by the administration of opium or kindred drugs.

Rankin and Palmer reported 58 cases of post-operative parotitis from The Mayo Clinic in which patients were not treated with radium, with 23 deaths, or a mortality of 39 per cent, and 20 cases in which patients were treated with radium, with four deaths, or a mortality of 20 per cent. Of these four patients who died following treatment with radium, only one died from acute parotitis alone, other complications not being present. Rankin and Palmer reached the following conclusions: (1) radium, if applied immediately on recognition of the infection, tends to abort suppuration and decrease mortality; (2) if surgical incision becomes necessary, the prognosis is more grave; (3) the extent of the infection, for example bilateral involvement, increases the mortality; (4) the more serious the operation preceding the infection, the higher is the mortality, as shown by studies of operations on the colon, and (5) the mechanism of the beneficial action of radium is unknown, but one possible theory is that the resultant disintegration of the protective leukocytic barrier liberates protective antibodies or ferments (5).

We have reviewed all cases of post-operative parotitis in which patients were treated with radium at The Mayo Clinic from 1929 to 1934, inclusive, or a total of 184 cases. This includes the 20 cases reported by Rankin and Palmer. The patients ranged in age from 7 to 76 years, the average age being 50; there were 95 males and 89 females. Table I gives the results of treatment according to the type of opera-

[1] Presented before the Radiological Society of North America, at the Twenty-first Annual Meeting, in Detroit, Dec. 2–6, 1935.

TABLE I.—RESULTS OF TREATMENT ACCORDING TO THE TYPE OF OPERATION FOLLOWING WHICH PAROTITIS DEVELOPED

Type of Operation	Cases	Per cent	Patients		Mortality, per cent
			Cured	Died	
Colon	62	33.7	41	21	33.9
Gall bladder	41	22.3	32	9	21.9
General abdominal	25	13.6	23	2	8.0
Pelvic	24	13.0	21	3	12.5
Partial gastrectomy	12	6.5	9	3	25.0
Kidney and bladder	10	5.4	6	4	40.0
Brain	3	1.6	3	0	0.0
Head and neck	3	1.6	3	0	0.0
Breast and thorax	2	1.1	2	0	0.0
Orthopedic	1	0.6	1	0	0.0
Prostatic punch	1	0.6	1	0	0.0
Total	184	100.0	142	42*	22.8

* This includes six patients who were moribund when treatment was begun, thus precluding completion of treatment. Hence, the true mortality is 36 patients, or 19.6 per cent. Of these, 11, or 6 per cent, died of parotitis alone, without other complications being present.

tion employed preceding the development of parotitis. Sixty-two cases, or 34 per cent of the total, represent operations on the colon (resection of the colon or rectum, colostomy, ileocolostomy, or enterostomy). Twenty-one of the patients died, a mortality of approximately 34 per cent. The only high death rate comparable to this was for the smaller group of operations on the kidney and urinary bladder. Pelvic surgery preceded the development of parotitis in only 13 per cent of the total cases, and the mortality was low (12.5 per cent). The great variety of the types of operations is of interest, even operations on the brain, radical operations on the breast, and orthopedic and prostatic punch operations being followed by the development of parotitis.

That extensive bilateral involvement is serious is demonstrated by the fact that, while only 32, or 17 per cent, of the total patients had bilateral acute parotitis, the mortality for this group was 40 per cent compared to a mortality of only 19 per cent for the remaining 152 patients.

TABLE II.—RESULTS ACCORDING TO INTERVAL BETWEEN DIAGNOSIS AND BEGINNING OF TREATMENT IN 122 CASES IN WHICH RECORDS WERE OBTAINED

Treatment Begun	Per cent	Cured	Died	Mortality, per cent
Within first 12 hours	90	82	28	25.4
Within second 12 hours	7	6	3	33.3
Second day	3	3	0	0.0

Of the total of 42 patients who died (including the six who were moribund and for whom treatment was abandoned) 11, or 6 per cent of the total of 184, died of parotitis alone, and 31, or 17 per cent, died of parotitis and other complications. These complications were multiple in five cases, and consisted of peritonitis in 11 cases, pneumonia in six, intestinal obstruction in two, uremia in one, pelvic infection in one, septicemia in one, cerebral hemorrhage in one, pulmonary embolism in one, hepatic insufficiency in one, and pulmonary edema in one.

The interval between the diagnosis of the infection and the application of radium has always appeared important. To forestall suppuration and lower the mortality, the earliest possible treatment has been stressed. We have regarded parotitis as an emergency, and have been prepared to carry radium to the bedside in any of the hospitals at any hour of the day or night such infection was reported. Some idea of the time interval for the whole group may be obtained from the bedside notes made in 122 cases (Table II). A slightly higher mortality attended delay in treatment beyond twelve hours. Too few were treated more than 24 hours after the onset of the condition to permit conclusions to be drawn. No patients were treated more than 48 hours after the onset of parotitis.

The technic of treatment consists in giving slightly more than 50 per cent of an erythema dose with radium surface blocks. A universal tube, containing about 50 millicuries of radon filtered through 2 mm.

TABLE III.—RESULTS ACCORDING TO WHETHER ONE OR TWO SETS OF RADIUM TREATMENT WERE EMPLOYED

Radium Treatment	Cases	Per cent	Patients		Mortality, per cent
			Cured	Died	
1 set	148	80.0	108	40	27.0
2 sets	36	20.0	34	2	5.5

of lead, as well as the usual 1 mm. of brass and 0.5 mm. of silver, is placed at a distance of 2.5 cm. from the skin on a wooden block. The base of the block is 3 cm. square; from two to four blocks are applied at a time. If the area of infiltration is too large to be covered with four blocks of this description, a second application of four similar blocks is placed around the periphery of the first. Each application is of eight hours' duration. Ordinarily, one set of treatments (consisting of these four or eight blocks) proves sufficient, and the gradual subsidence of the swelling takes place during the next three or four days. However, in cases in which there is no apparent regression of the swelling after several days, or in which there is an actual increase in the size of the lesion, a second set of blocks may be applied over the same areas after the completion of the previous treatment, but the time is shortened to six hours. Analysis of the results according to whether one set was sufficient or whether two sets were required is shown in Table III. In the 36 cases in which a second application was necessary, the results were unusually good.

The toughness of the capsule of the parotid gland makes fluctuation a rather late sign in abscess formation; the infection may rupture spontaneously before fluctuation becomes evident. In Table IV it will be noted that in six cases spontaneous rupture occurred, with subsequent recovery. When fluctuation is evident, surgical incision and drainage should be instituted; in three such cases in which drainage was not instituted, only one patient recovered. In six cases in which drainage was secured, recovery occurred in four. As a supplementary treatment we advocate warm moist dressings, as intense dry heat or ice packs superimposed on the areas receiving radium treatment may be detrimental to the skin.

TABLE IV.—RESULTS OF RADIUM TREATMENT ACCORDING TO SUPPLEMENTARY MEASURES EMPLOYED

Supplementary Treatment of Parotitis	Cases	Patients		Mortality, per cent
		Cured	Died	
Incision and drainage	6	4	2	33.3
Spontaneous rupture	6	6	0	0.0
Fluctuation present; not drained	3	1	2	66.6

In summary, over a six-year period we have treated 184 patients with radium packs for acute parotitis following major operations. The mortality, grossly, was 22.8 per cent, but discounting the six deaths of moribund patients which occurred before treatment could be more than started, the true mortality was 19.6 per cent. This compares with the mortality of 58 per cent in the combined American statistics, quoted by Green. Most of these deaths are due to parotitis plus other serious complications; in our series only 6 per cent died of parotitis alone. The conclusions reached in our series of 184 cases substantiate those of Rankin and Palmer for their group of 20, in which patients were treated with radium. The largest incidence and approximately the greatest mortality follow colonic surgery; the necessary withholding of food and fluids by mouth for several days following operation and the effect of the opiates required and the absorption from intestinal stasis may all play a rôle. The more serious the preceding operation, the higher the mortality following the development of parotitis. Practically any surgical procedure may be followed by acute parotitis, the condition usually being associated with other complications. Bilateral parotitis is fortunately not common and occurred in only 17

per cent of the cases in our series, but when it did occur the mortality was doubled. Surgical incision and drainage should be carried out when definite fluctuation is noted.

From our clinical experience with these patients, we believe that radium should be applied at the earliest possible moment after the diagnosis of parotitis is made. Only a moderate dosage is necessary. The only supplementary treatment needed is warm moist dressings or packs and general nursing care. Within a few days the swelling gradually subsides and the condition is cured. If regression does not occur in a few days, or the swelling increases, a second application of radium over the same areas, at lesser dosage, is often successful. Suppuration is usually aborted by radium treatment, but when it does occur, surgical incision and drainage is indicated. We have seen no detrimental effects from treatment as described above, and there are no contra-indications for radium treatment of this seriously ill group of patients.

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THE PRESENT STATUS OF THE X-RAY AS AN AID IN THE TREATMENT OF GAS GANGRENE[1]

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IN December, 1931, just four years ago, we reported before this Society the use of the x-ray as an aid in the treatment of a few cases of gas gangrene.

In all, eleven cases were reported, eight of which received x-ray treatment. In six cases in which the extremities were involved, all recovered, and in two cases in which the trunk was involved, both patients died. There were three other cases reported which received no x-ray treatment: two of these patients died, while the one who lived, a six-year-old child, lost his arm at the shoulder from an infection at the elbow. In none of the six cases with extremity involvement treated with x-ray was it necessary to amputate a limb because of the infection, so it appeared that x-ray not only cut down the mortality but also saved the extremities.

The first report and its major conclusions will be reviewed here briefly. Although this first series was extremely small, the comparative results of the six extremity cases treated with x-ray with the three cases which were not so treated was so strikingly in favor of adding x-ray to the other measures in use that we thought we should present the matter, hoping that others might try it.

This hope was not entirely unfounded and the method has received some trial by others during the last four years. Through personal communications and from the cases reported in the literature we have collected data on 40 cases. Some of the features connected with the treatment—complications and results of these 40 cases in which the x-ray has been used more or less—form the basis for the present report.

The data on these cases will necessarily be brief.

At the time of our first report we used the following technic, but recommended that for extremity work in the future, a heavier dosage be given.

TABLE I.—ORIGINAL TECHNIC

Mobile x-ray unit: 5–30 radiator type of tube.
5-inch spark gap (approximately).
5 milliamperes.
15-inch distance (approximately).
0.5 millimeter aluminum filter.
3 minutes morning and evening for 3 days.

This type of apparatus is found in almost any hamlet or village, and the above directions for its use are simple and safe. As this amount of radiation was very small, it seemed almost incredible that any dose, such as recommended in Table I, would have any therapeutic effect, but the facts as observed were reported with the request that others try it, giving greater dosage in the trunk cases. We also requested that none of the usual methods of treatment be neglected, as it was not necessary to do so in order to use the x-rays.

We intend to recommend a definite change on this latter point in our summary to-day.

TABLE II.—CASES FROM ORIGINAL SERIES WHICH HAD X-RAY AND SERUM

Age	Extrem.	Trunk	Amput.	Living	Dead
21	Yes	No	No	Yes	
10	Yes	No	No	Yes	
23	Yes	No	No	Yes	
42	Yes	No	Yes	Yes	
61	Yes	No	Yes (2)	Yes	
8	Yes	No	No	Yes	
55	No	Yes	No		Yes
47	No	Yes	No		Yes
Totals[2]	6	2	2	6	2

Mortality in extremity cases, none.
Mortality in trunk cases, 2 (100 per cent).
Mortality in series, 25 per cent.

[1] Read before the Radiological Society of North America, at the Twenty-first Annual Meeting, at Detroit, Dec. 2–6, 1935.

[2] Cases not receiving x-ray are omitted. Total cases, 3; deaths, 2; arm amputation, 1; mortality in untreated cases, 66 per cent.

Comment.—In this series (see Table II), it appeared that the extremity cases were definitely benefited by adding x-ray to the other methods of therapy.

In the trunk cases, due to increased density of the parts involved, it was suggested that more filter and higher voltage be used. This short series is reported because of the seriousness of the infection, and it was thought best not to wait until a greater number accumulated. Since that time we have gathered information on 40 cases. They are tabulated in Table III.

It would appear from Table III that two patients died of unavoidable post-operative accidents (sloughed ligature and embolism), and the third patient from diabetic complications which were hardly surgical at time of admission. One patient died on the third day after admission to the hospital; he had received but very little x-ray treatment, had an amputation, and shock was undoubtedly a factor in his death. The other case received only a prophylactic treatment and no x-ray during the course of the disease—obviously under-treated.

Upon dividing these cases into extremity and trunk groups, we find that all the trunk cases recovered in the second series and all the mortality in the second series was in the extremity cases. This was the complete reversal of our findings in the first series.

Upon further analysis of the extremity cases in the second series we find that there were 28 cases; 17 were not operated on (and by "no operation" is meant that no extremity was amputated), 11 were operated on, losing an extremity, and among this 11 we find our five dead. Table IV shows comparative figures on these points in the two series.

TABLE IV.—COMPARATIVE MORTALITY IN TRUNK AND EXTREMITY CASES

(*Both Series*)

	No. Cases	Dead	Percentage Dead
Trunk			
First Series	2	2	100
Second Series	8	0	0
Extremity			
First Series	6	0	0
Second Series	28	5	18

Further analysis of the second series—extremities only—brings to light the following facts in their treatment.

TABLE III.—PRESENT SERIES FROM SEVERAL SOURCES, INCLUDING CASES IN ORIGINAL SERIES

	Total	Serum	X-ray	Extrem.	Amput.	Trunk and Extrem.	Living	Dead of G. G.	Dead of Comp.	Remarks
Dr. Cogley	5	5	5	5	0	0	5	0	0	
Dr. Condon and Dr. Newell	2	1	2	2	1	0	2	0	0	One case; no serum; recovered
Dr. Faust	9	8	9	7	1	4	9	0	0	One case; no serum; recovered
Dr. Fouts	2	2	2	2	1	0	1	0	1	Ligature slipped, ninth day; dead in two hours
Dr. Hanchett	2	2	2	2	1	0	2	0	0	
Dr. Hunt, Dr. Johnson, and Dr. Bisgard	7	7	7*	7	5	2	4	2	1	Diabetes mellitus; died on third day from bronchopneumonia
Dr. Kelly	10	10	10	7	2	3	8	2	0	
Dr. Merritt	1	1	1	1	1	0	1	0	0	
Dr. Stech	1	1	1	1	1	0	0	0	1	Pulmonary embolism after repair of stump, twenty-third day
Dr. Turner	1	1	1	0	0	1	1	0	0	
Totals	40	38	40?	34	13	10	33	4	3	

* Patient received prophylactic x-radiation, not treated during the active course of the disease. He died eight hours after amputation on the fifth day.

Percentage mortality of cases dead of gas gangrene, 10 per cent. Percentage mortality of all cases in series, 17 per cent.

COMPARATIVE MORTALITY—CASES OPERATED ON AND NOT OPERATED ON

(*Second Series*)

	No. Cases	Dead	Percentage Dead
Extremities only	28	5	18
Cases not operated upon	17	0	0
Cases operated upon (amputation)	11	5	45

Investigation of the records of the five patients who died brings to light the following facts:

1. The man who died two and a half hours after the ligature sloughed off a large vessel in the stump on the ninth day, had apparently recovered from gas gangrene.

2. The man who died of a pulmonary embolism on the twenty-third day—the embolism followed some repair work on the stump—also had evidently recovered from gas gangrene.

3. The man who died on the third day after admission, who received two x-ray treatments but failed to rally after an amputation, had been severely injured.

4. The man who received 100 r the first day and 90 r the second day of his disease and no further x-ray, died the fifth day, eight hours after an amputation. (Treatment should be continued vigorously for at least three days.)

5. The man on whom the admittance diagnosis was diabetes with impending coma, had a gas bacillus infection in a diabetic ulcer. He died on the third day following an amputation, after having received two doses of x-ray. This was not sufficient treatment. Postmortem showed extensive bronchopneumonia.

For the sake of comparison we are including some figures on the mortality rate of cases treated without x-ray (Table V).

The cases shown in Table V indicate a mortality percentage of 39.4. In the 40 cases here reported which received x-ray in addition to the other measures, we show a 17 per cent mortality rate if all cases are included. We show only a 10 per cent mortality rate if the three patients who are thought to have died from causes other than gas gangrene are excluded, and in the 21 cases involving extremities, that received only local surgery and no amputations, we show a 0 per cent mortality.

TABLE V

Name	No. Cases	Percentage Mortality	Date	Ref.	Remarks
Hanchett quoting Millar	607	49.7	1932	West. Jour. Surg., April, 1935, quoted by Hanchett	Cases from civil life
Hanchett quoting A. E. F. statistics	Not stated	48.52		West. Jour. Surg., April, 1935, quoted by Hanchett	A.E.F.
Elliot Easton	17	20	1935	Ann. Surg., June, 1935	Cases from civil life

CONCLUSIONS

If one may be permitted to draw conclusions from such a small series of cases, it seems to be definitely certain that x-ray treatment is indicated in gas gangrene, both in extremity and in trunk cases; that the treatment should be started as soon as the disease is suspected and be given throughout its course, twice each day for at least three days.

It seems that amputation is definitely detrimental to the welfare of the patient having gas gangrene; if not detrimental, at least unnecessary, and if unnecessary, who wants to throw an arm away? Often these patients are children—one child 11 and one 12 years of age in this second series recovered after losing an arm and both had active gas gangrene above the site of amputation shortly after the amputation was finished, so all the disease was not eradicated by the surgical procedure

Do not be discouraged and think the patient is beyond hope when you are first called upon to treat him, as this work was started on material of that type, when the surgeon had his back to the wall, so to speak, when the case looked hopeless; in other words, the type of case all radiologists are quite familiar with in the inflammatory group.

It appears, therefore, that it would be safe to rely entirely upon the serum, local

antiseptics, local minor surgery, and x-ray, since the five deaths in this series in the extremity cases all had amputation of an extremity, while all those having no amputation survived. Of the 13 cases operated upon (amputation), five died and eight recovered, while in the remaining 21 cases not operated upon, with extremity involvement, none died. The entire non-amputated group recovered, relying entirely upon serum, local antiseptics, and small doses of x-ray given frequently.

Since many of the cases which had radical surgery still had infected tissue left behind, it raises the question as to what good the surgery really does. Then the mortality of the cases operated upon is so terribly high as compared to the cases not operated upon, that again the use of amputation seems to be a distinct disadvantage.

In two cases in this series no gas bacillus serum was used and both cases recovered, though one of them developed tetanus after the gas gangrene had subsided. The patient also recovered from the tetanus through the use of large doses of anti-tetanic serum. This latter case should serve as a distinct warning against the omission of anti-tetanic serum.

Finally, the procedure is based entirely upon clinical evidence and more work must be done before the exact status of the use of the x-ray in this condition is determined. However, some work done at Stanford University (1) on radiation reaction in living tissue leads to the conclusions that, following radiation (H_2O_2), hydrogen peroxide is formed in the tissue radiated. If this is true and since we all know these organisms are anaerobic, this formation of hydrogen peroxide may be the answer to the problem. At any rate, we believe that (1) x-ray treatments are indicated in all cases of gas gangrene; (2) treatments should be started regardless of how hopeless the case may seem; (3) treatments should be given morning and evening or twice daily and of sufficient voltage to insure penetration of the involved tissue—about 90 to 100 kv. on the extremities, from 130 to 160 kv. on the trunk, and about 100 r units per dose. Finally, we believe that one should discourage amputations except in those instances in which the tissues are hopelessly damaged by causes other than gas gangrene. The dark appearance of the involved tissues does not indicate a true gangrene, and it clears up following successful radiation. It is probably localized cyanosis.

The essayist wishes to thank Dr. J. J. Faust, of Tyler, Texas; Dr. J. Philip Cogley, Dr. McMicken Hanchett, and Dr. J. L. Stech, of Council Bluffs, Iowa; Dr. Albert P. Condon, Dr. Charles Newell, Dr. Howard B. Hunt, Dr. H. F. Johnson, Dr. J. Dewey Bisgard, and Dr. Roy W. Fouts, of Omaha, Nebraska; Dr. E. A. Merritt, of Washington, D. C., and Dr. B. Weems Turner, of Houston, Texas, for permission to include their cases and the assistance given through correspondence.

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ROENTGEN THERAPY IN THE TREATMENT OF NON-SPECIFIC RESPIRATORY DISEASES[1]

By A. JUSTIN WILLIAMS, M.D., *and* LLOYD BRYAN, M.D., *San Francisco*

ROENTGEN therapy of non-specific infectious disease of the respiratory tract is not new. The action is not specific, but is undoubtedly the same as in other inflammatory processes.

In the following series there has been no selection of cases; subjects were recruited wherever possible. An attempt was made to run a control series, but this failed because it was impossible to secure the cooperation of the control cases.

The results reported are based chiefly on the subjective symptoms of the patients. An attempt was made to follow the cases carefully by clinical examination and roentgenograms, but this was found to be unsatisfactory since many patients would not return after clinical symptoms subsided. However, a sufficient number of cases were followed in such a manner to warrant the assumption that the final results were essentially the same in either case. Many cases followed as long as two years have had not only relief from the current infection but have reported relative freedom from respiratory infections since the treatment. So far as known there have been no unpleasant results following treatment which could be attributed to radiation. No case has shown any visible skin change or any other undesirable effect. Cases treated were either benefited or ran their usual course, with the exception of cases of bronchitis and tracheitis which will be discussed later. All patients are warned before treatment is started that they may expect a transient exacerbation of symptoms; otherwise, many become dissatisfied and fail to return for further treatment.

RHINITIS

After a few cases early in the series the dose applied has been 20 r daily. The results were identical, using either high or low voltage and 0.5 mm. Cu or 0.25 mm. Cu plus aluminum filtration. Larger doses, especially in the very acute cases, caused an increase in the nasal discharge and obstruction, coming on in from six to twelve hours and lasting up to thirty-six hours after the first treatment. This occurrence resulted in many "incomplete" cases. In addition to an increase of symptoms, distressing dryness of the nasal passages followed the use of larger doses. Dosages of 40, 60, 80, and 100 r were tried but soon discarded. Using doses of 20 r, about 8 per cent of the cases complained of transient increase in symptoms coming on in six hours and lasting up to twenty-four hours.

Treatments were given through a field measuring 8 × 11 cm., sufficient to cover the sinuses and nasal passages. The eyes were covered by lead goggles bridged by tape to allow exposure of the ethmoid cells. The latter is perhaps unnecessary, but with the goggles one can be certain that there is no possibility of epilation or conjunctival reaction.

ACUTE RHINITIS, 170 CASES:
SUMMARY OF TREATMENT

Duration		*Number Treatments per Case.*				*20 r per Treatment*
		1	*2*	*3*	*4*	
Day of onset	42	12	20	6	4	
2nd day	56	6	40	8	2	
3rd day	40	4	28	6	2	
4th day	12		10			
5th day	4		4			
6th day	4		4			
7th day	12		12			
		11.8%	69.2%	13%	6%	

ACUTE RHINITIS, 170 CASES:
SUMMARY OF RESULTS

From First Treatment	*Symptom-free, %*	*Improved, %*	*No Change, %*
24 hours	12	78	10
48 hours	56		
72 hours	19		
Total	149-87	13-8	8-5
96 hours	5	10	6

21 cases apparently unaffected.
28 increased nasal discharge.
6–12 hours after treatment. Final result not affected.

[1] Presented before the Radiological Society of North America, at the Twenty-first Annual Meeting, in Detroit, Dec. 2–6, 1935.

SINUSITIS

Acute cases were treated daily and chronic cases on alternate days. An initial dose of 80 r was given, and later the dose increased up to 150 r if necessary. Many patients complained of increased headache following the larger doses. This is avoided in most instances when the smaller dose is employed, but does occur in a small percentage of cases, especially in cases in which headache is a prominent symptom of the disease.

Many of the patients who were benefited by the treatments had increased nasal discharge within forty-eight hours following the initial treatment. This is regarded as a good prognostic sign, but did not occur in all cases in which good results were obtained. The most striking results were noted in those cases with good drainage; many such cases had had intranasal surgery. Roentgenograms in many cases demonstrated clearing of dense sinuses following treatment. No contraindication to irradiation of sinusitis has been noted.

SINUSITIS, 88 CASES: ACUTE AND CHRONIC

History from 3 days to 2 years.
52 cases treated daily, av. 3.4 treatments each.
36 cases alternate days, av. 4.5 treatments each.

From First Treatment	*Symptom-free, %*	*Improved, %*	*No Change, %*
1 day	0	67	33
2 days	18	59	23
3 days	24	43	15
4 days	17	27	12.5
5 days	8	20	
7 days	2.5	18	
9 days	2.5	16	
11 days	3	12.5	
14 days		12.5	12.5
Total	66–75	11–12.5	11–12.5

BRONCHITIS

Acute cases were treated daily and chronic cases on alternate days. Usually the entire chest was treated through one large field, alternating anteriorly and posteriorly. Those with a tracheitis, having a dry unproductive cough originating with a tickling sensation in the throat, have accounted for a majority of the failures. Many of these cases were made worse by irradiation, probably because of the drying effect on the mucous membrane. At present, in such cases each side of the chest is treated, shielding the mid-line with lead to protect the trachea from direct exposure. The results have been more satisfying since this procedure has been adopted. Dosage of from 80 to 120, depending upon the size of the patient, have proven to be as effective as larger doses.

In both the acute and chronic types, many patients had increased symptoms, with loose cough and increased sputum, beginning as early as twelve hours after the first treatment. This is regarded as a good prognostic sign.

Cases of combined upper and lower respiratory infections have been treated, using the same technic and dosage as for the individual lesions. The results obtained were comparable with those with localized disease.

BRONCHITIS, 53 CASES: BOTH ACUTE AND CHRONIC

History of disease from 1 day to 6 months.
3 cases received 1 treatment daily.
25 treated daily, average 2.4 treatments each.
25 alternate days, average 6.7 treatments each.

From First Treatment	*Symptom-free, %*	*Improved, %*	*No Change, %*
1 day		57	43
2 days	15	49	40
3 days	19	40	17
4 days	13	40	13
5 days	19	24	9
7 days	1.5	23	9
9 days	4	19	9
10 days	4	14	9
12 days	1.5	13	9
14 days	6	7	9
Total	44–83	4–7	5–9

TOTAL SUMMARY

	Total	*Symptom-free, %*	*Improved, %*	*No Change, %*
Bronchitis	53	44–83	4– 7	5– 9
Rhinitis	170	149–87	13– 8	8– 5
Sinusitis	88	66–75	11–12.5	11–12.5

Incomplete cases, results not known, 78.

THE BLOOD PICTURE IN X-RAY AND RADIUM WORKERS[1]

By IRA I. KAPLAN, B.Sc., M.D., Director, Radiation Therapy Department, *and* SIDNEY RUBENFELD, B.Sc., M.D., Assistant Visiting Radiation Therapist, Bellevue Hospital, *New York City*

NOT long after the roentgen ray and radio-active substances were introduced into the field of medical therapeusis, their deleterious effect upon the blood picture was noted. This constitutional effect, however, was entirely dissociated from the local destructive action due to over-exposure or improper filtration. Fortunately, the warning was heeded and methods were devised to protect the workers in the new science.

Heineke (3), in 1904, introduced experimental evidence of the effects of radiation when he noted a marked leukopenia and little or no alteration in the red cell count. Very similar findings, but with an additional conclusion that the lymphocytes were the most sensitive elements, were reported by Helber and Linser (4), in 1905. The following year a contribution by Benjamin, Reuss, Sluka, and Schwartz (2) revealed the production by x-rays of a transient polymorphonuclear leukocytosis. These experimental data were given great impetus and assumed clinical importance when, in 1911, von Jagie, Schwartz, and von Siebenrock (12) reported four cases of lymphatic leukemia occurring in x-ray workers. They attributed the malady to irradiation received over a prolonged period. Following upon this report, investigations were undertaken upon the personnel of the various staffs.

Aubertin (1), in 1912, presented his analysis of 16 radiologists. In these, there were two groups which manifested different effects; one group of six cases showed a leukopenia with a decrease in the polymorphonuclears, and the second group presented a mild increase in polymorphonuclears and eosinophiles. He was unable to detect any abnormal forms in the blood. H. Béclère, adding a discussion to Aubertin's paper, stated as his belief that there was not any significant alteration in the blood count status; leukopenia and eosinophilia were his occasional findings.

The first American report appeared in 1922, when Pfahler (10) gave a comprehensive review of observations in many clinics throughout the country. The conclusion drawn after a survey of 338 returned questionnaires was that only after undue exposure did there occur a leukopenia with a relative lymphocytosis, and only occasionally was an eosinophilia noted.

In 1925, Portis (11) submitted the results of his analysis, showing the most evident alteration to be a progressive diminution in the white components, due to a lower polymorphonuclear count, while the lymphocyte count remained stationary or elevated slightly. No change occurred in the red cell or hemoglobin status.

Lavedan (6), in 1927, presented the results of a very exhaustive review in which was revealed the frequent, and what he believed to be the characteristic, occurrence of an absolute lymphocytosis, due to an actual increase in the mononuclear elements. Less rarely, there appeared a relative increase in cases in which the polymorphonuclears increased and the lymphocytes remained at the normal level. He emphasized the rapidity with which it may appear, but made light of its seriousness, especially since abstention from work always corrected the change.

Mottram (7) reported a diminution in the total red cell count in these workers. In another investigation, Mottram (8) showed, by experiments on animals, that the bone marrow is less affected by x-rays than are the lymphoid tissues. Mottram and Clarke (9) reported a clinical survey in which two groups of workers were studied.

[1] Presented before the Radiological Society of North America, at the Twenty-first Annual Meeting, in Detroit, Dec. 2–6, 1935.

viz., radium technicians and radium clinicians, and in which both groups revealed a lower level of the polymorphonuclears and lymphocytes compared with normal counts.

SCOPE OF THE SURVEY

We have been afforded the opportunity to observe a group of workers, both technicians and clinicians, over a period of from one to four years. Realizing that the type of radiation manipulated, and the environmental surroundings are the common denominators, we have divided this series into: Group I, those doing x-ray diagnosis; Group II, radium element technicians; Group III, radium emanation technicians; Group IV, radium and x-ray therapy technicians, and Group V, radium pack nurses. The blood counts of these individuals were taken with varying degrees of regularity during the years, but nevertheless offered adequate data for this analysis. In each group, there was a uniform regularity as to the time of day when the blood was examined. Thus, the workers in Group III were examined late in the afternoon, and those in Group IV, late in the morning, before lunch.

In order to correlate the blood picture with the quantity of stray radiation received by the technicians, the following procedure was adopted. The technicians in each of several groups were required to carry a dental film in the chest pocket of the uniform or jacket. After a period of from 12 to 14 days, these films were turned over to our physicists, Messrs. Braestrup and Blatz, for analysis. The evaluation of the films was obtained by comparison with films exposed to known quantities of radiation. All the films were developed and fixed together, taking the usual precautions to insure uniform processing. Upon completed dark room procedure, the blackness or density of all the films was determined on a photo-electric cell densitometer designed for this particular purpose. A graph was made showing the relationship between the blackness and the corresponding quantity of radiation. From this curve, the dosage received by the films carried in the pockets of the technicians was determined. While this method is subject to the objection that the quality of stray radiation may differ from that used as a standard, this is not serious, since we are mainly interested in the relative values of stray radiation.

TABLE I

Group	Average Daily Exposure	Percentage Transmitted through Lead Foil	
		Thin	Thick
X-ray diagnosis	0.012 r	0%	0%
Radium pack	0.038	82%	74%
Radium and x-ray	0.034	33%	14%

In order to distinguish between the stray radiation produced by radium and x-rays, a series of tests were carried out, using several films on which parts were covered with different thicknesses of lead foil. It was found, as may be expected, that the technicians manipulating radium exclusively received stray radiation of much greater penetration. (See Table I.) The soft ray emitted from the x-ray diagnostic tube failed to penetrate either thickness of the lead foil. The gamma ray from the radium pack was sufficiently hard to strike the film and produce a distinguishable darkening.

It was evident from these tests that our means of protection were adequate.

Group I. X-ray Diagnosis.—Particular significance was attached to this group, because it included x-ray technicians in Bellevue Hospital, where the amount of plate work is tremendous—probably not surpassed in any other institution. It has been estimated that some five hundred films are taken daily. From the rather large group of workers, we chose four individuals as representative of the different routines performed. Upon a reading of their blood reports it was seen that the hemoglobin, red cell, and white cell counts, although exhibiting slight variations from time to time, hovered more or less about the normal level. More definitely, in no single case did a decrease in these elements appear. The polymorphonuclear and lymphocyte ratio remained unaltered in all but one

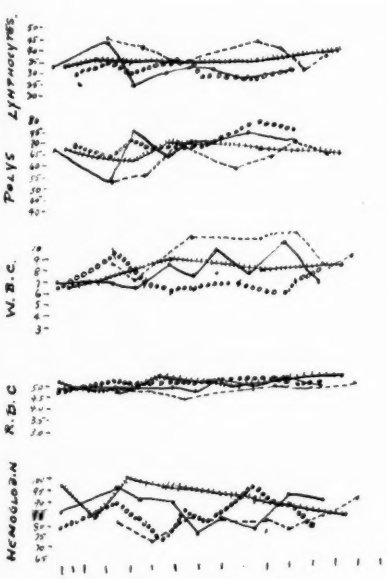

Chart I. X-ray diagnosis.

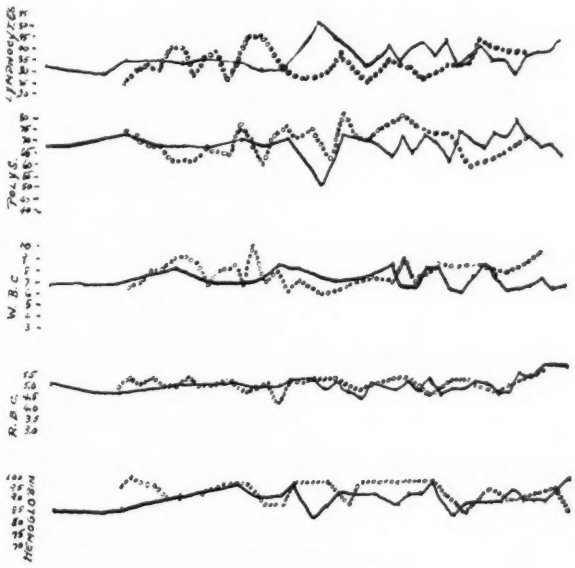

Chart II. Radium element.

case, in which there was a persistently low polymorphonuclear and high lymphocyte count. As an instance, over successive months, the lymphocytes read 42, 40, 33, 42, 38, 29, 35, indicating that increased precautionary measures failed to correct the changed ratio. The assumption is, therefore, that this individual was more sensitive to the amount of stray radiation received, and that the normal was maintained at a different level. The decrease in polymorphonuclears was always compensated for by an increase in lymphocytes. Eosinophilia was not characteristic. (See Chart I.)

Group II. Radium Element Technicians.—Observations were made over a period of five years on two workers engaged in radium preparations for a commercial organization, where from 3 to 5 grams of radium are manipulated per month. Ample protection is provided, and the working environment is the same for the individuals. Analysis of the counts shows that the hemoglobin and red cell elements have definitely been unaltered over this period. The total leukocyte reading of one has not deviated, while that of the second worker has approached a lower level in the past two years, remaining at that stratum, although never lowering sufficiently to be classified as a leukopenia. The polymorphonuclear-lymphocyte ratio in one individual was rarely disturbed in five years; there were but three instances in which the lymphocytes rose above 35 per cent. Similarly, the second worker manifested infrequent alterations in the white components, but a noteworthy finding here was the generally higher level of the lymphocytes, constantly hovering about the high normal reading. Again, as with the x-ray diagnosis technician, we suggest this factor as evidence of the greater sensitivity of the individual. Precautions were often emphasized whenever a change in the P-L ratio was observed, and the decrease in lymphocytes was usually rapid. As an instance:

June 10:	Polys.	48%
	Lymphocytes	48%
March 12:	Polys.	58%
	Lymphocytes	40%
	and again	
July 16:	Polys.	70%
	Lymphocytes	30%
April 18:	Polys.	70%
	Lymphocytes	28%

(See Chart II)

Group III. Radium Emanation Technicians.—The opportunity was afforded to

study two groups of workers preparing radium emanation, one in a commercial laboratory and the other in a hospital emanation plant. The latter three technicians, working in a common environment, manifested some striking variations in reactions. As in other groups, the hemoglobin and red cell count remained at normal readings. The total leukocyte count was constantly maintained at lower levels, often about 4,000. As blood examinations were instituted after they had been employed for several years, it cannot be estimated whether there was a progressive decline, or whether that level was within the normal range for the two individuals in whom this diminution occurred.

The ratio of the white cell components was frequently disturbed, always characterized by a decrease in the polymorphonuclears and an increase in the lymphocytes, the latter being mature forms. Eosinophilia did not appear, and there was no predominance of transitional forms. Of special interest, the low leukocyte count occurred simultaneously with the alteration in the P-L ratio. Following a vacation, return to the normal was often evident. For instance, one technician, on July 31, 1934, had a total white count of 3,900, with 49 per cent polymorphonuclears and 49 per cent lymphocytes. The count on Aug. 16, 1934, showed the white cells to be 4,400 and 70 per cent polymorphonuclears, with 27 per cent lymphocytes. (See Chart III-*a*.)

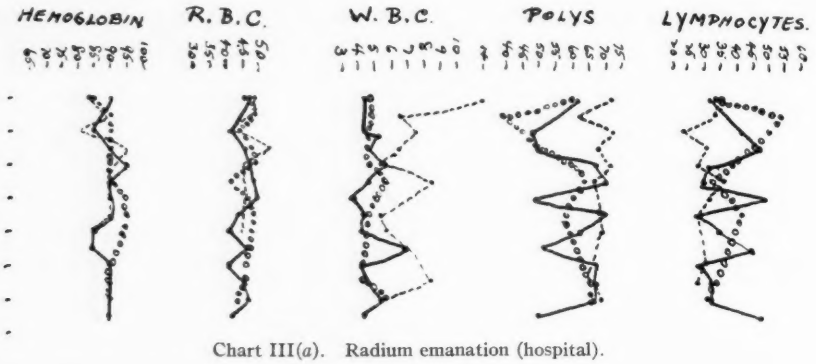

Chart III(*a*). Radium emanation (hospital).

Five individuals preparing radium emanation for a commercial organization were studied. Blood readings were done over a period of from four to seven years, often at regular monthly intervals. There were no instances of a decrease in the hemoglobin or red cell count. Unlike the previously analyzed hospital group, there did not appear a diminution in the total white count. Reactions were manifested by an alteration in the white elements in which the polymorphonuclears decreased and the lymphocytes increased. Only rarely did successive months reveal the deleterious effect. Instances of white cell change were recorded once in three years for W. B.; 12 times in six years for S. S.; 9 times over a period of six years for G. E.; 7 deviations in eight years for R. L. One technician, however, displayed numerous counts in which the polymorphonuclears were diminished, and the lymphocytes correspondingly elevated along with an eosinophilia of from 5 to 8 cells. In all the workers save one (A. K.), over the years studied, two successive months revealed a return to the normal level at the second reading. In the exception (A. K.), there were several instances in which the lymphocytes persisted at an elevated level for two or three months. (See Chart III-*b*.)

Group IV. Radium and X-ray Therapy Technicians.—Four technicians who manipulated deep therapy machines and radium element applicators were available for study, with blood readings obtained

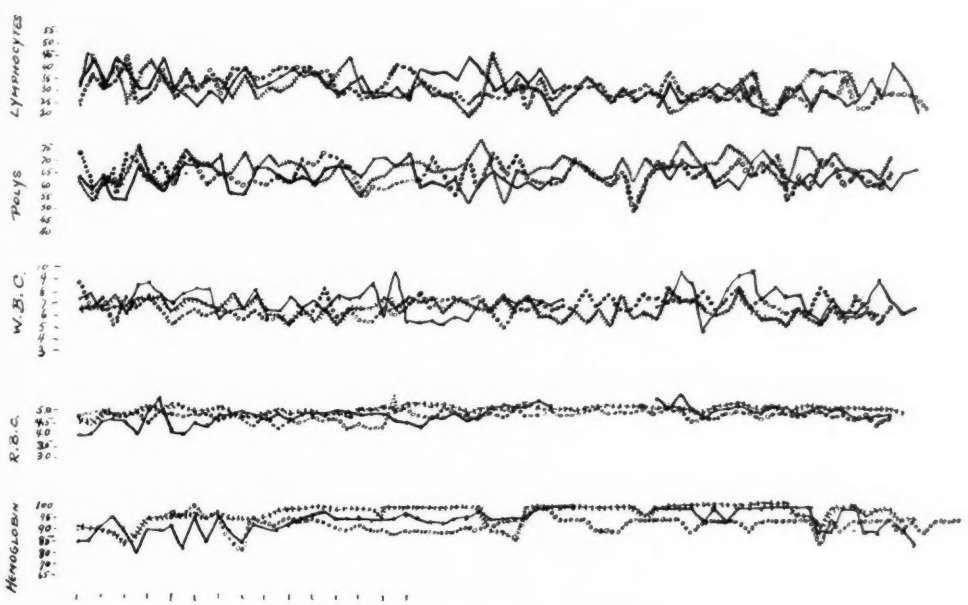

Chart III (*b*). Radium emanation (commercial).

over a period of three years. Environmental conditions were similar, and a plan of alternation was followed so that during the month each was exposed to approximately equal amounts of stray rays. That this condition obtained, was definitely corroborated by the physical findings.

Hemoglobin variations were slight, and at no time was a diminution noted. On the other hand, the one individual (C. S.), who showed occasional decreases in the red cell count below normal limits, revealed a gradual but progressive elevation in hemoglobin from 65 per cent, in 1932, to 85 per cent, in 1935. On five readings during these three years, the red cell count was below four millions, but, interestingly, the leukocyte components did not alter at the times when the red cell diminution was dominant. On seven readings, the P-L ratio changed, characterized by a decrease in the former and an increase in the latter. There was no concomitant reduction in total leukocytes. Moreover, frequently the eosinophiles were as numerous as from 5 to 8 per 100 cells, the probable explanation for which is an attack of malaria in youth. A second worker manifested frequent diminutions in the leukocytes with simultaneous alterations in the polymorphonuclear-lymphocyte ratio. A lymphocyte count of 40 per cent or more became a rather constant finding. Judging from this information, blood damage apparently was done, but the genital system was unaffected, since she was married, and delivered a normal, full-term fetus one year after cessation from work. Two other technicians exhibited no changes of any consequence. (See Chart IV.)

Group V. Radium Pack Technicians.—Four nurses who manipulated the 5-gram radium pack at Bellevue Hospital were included in this analysis. The number of blood readings for each individual is not many, yet some very definite effects were noted and are worthy of comment. There was no great variation or reduction in the hemoglobin content. An anemia of four months' duration, with a red cell count below four million, was evident in one nurse, while the three others showed no red cell

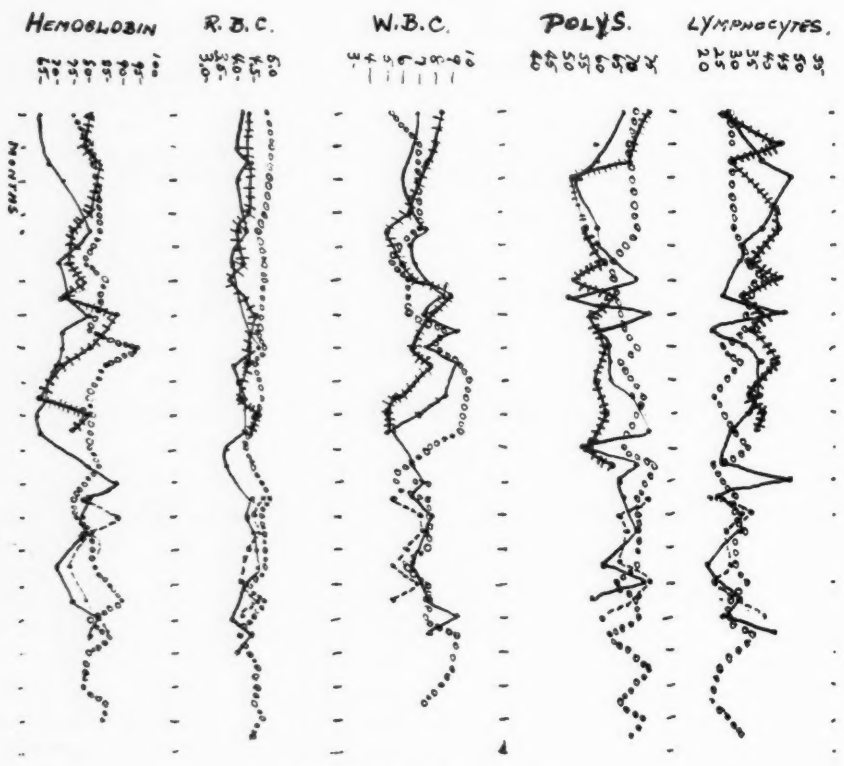

Chart IV. Radium and x-ray.

count variations. The nurse who showed the red cell anemia had a complete inversion of the polymorphonuclear-lymphocyte ratio when, on one occasion, the red cells were 3,400,000, the leukocytes 5,300, with 42 per cent polymorphonuclears, and 58 per cent lymphocytes. We deemed it best to change the service of this nurse, and subsequent readings, after several months, disclosed an approach toward the normal. Two other technicians exhibited a change in the P-L ratio quite frequently. One nurse manifested no abnormal reactions during a period of six months. (See Table II.)

SUMMARY AND DISCUSSION

In 1934, the International and British Protection Committees recommended that "under satisfactory working conditions, a person in normal health can tolerate exposure to x-rays to an extent of about 0.2 r per day." Whenever such physical determinations were feasible, in Groups I, III, and IV, we found exposures far below this tolerance dose. The determinations with two thicknesses of lead proved interesting, inasmuch as the stray radiations from the x-ray diagnostic machines did not penetrate any layer of the metal. On the other hand, the rays from the radium pack were obviously more penetrating, and about 74 per cent of the total was recorded through the heavier thickness, and approximately 82 per cent came through the thin layer.

Technicians working with the x-ray diagnostic machines manifested little or no reactions. One individual, with a decrease in polymorphonuclears and an increase in lymphocytes, failed to rectify this alteration even after greater precautions, vacation, or change of routine.

The workers with radium element showed no deviation in the hemoglobin, red cell count, or white cell count. One individual presented continuous readings of a high level lymphocyte, which probably established itself as the normal for that person. These technicians showed a rapid return to the normal after an altered reading.

Although the hospital radium emanation technicians revealed frequent white cell variations, the hemoglobin and red cell count were not reduced. This group stands as the one in which a low white count approaching a leukopenia, with a relative lymphocytosis, was a fairly constant finding. Aubertin, Portis, and Pfahler recorded similar alterations. In this group, return to normal after a vacation was the rule.

No hemoglobin or red cell diminution was observed in the technicians preparing commercial radium emanation. Unlike the hospital group, the total white cell level was maintained, but a change in the white components was a common finding. In only one individual was the alteration present in successive months.

Of the four technicians who manipulated both deep therapy machines and radium applicators, two revealed changes while two remained unaffected. The blood changes were demonstrated by a decrease in the polymorphonuclears and an increase in the lymphocytes, an occasional decrease in total leukocytes, and a sporadic eosinophilia. An old malaria may well explain the latter finding in one individual.

The one instance in this survey of a red cell anemia appeared in a nurse who manipulated the 5-gram radium pack. Determination of her exposure revealed a level below the tolerance dose of 0.2 r. This same individual was the only one to show a complete inversion of the polymorphonuclear and lymphocyte ratio. The outstanding record in the other workers was a decrease in the polymorphonuclears and an increase in the lymphocytes, without any other changes. A fact that deserves especial emphasis is that these changes appeared soon after commencing routine in the new field.

None of the 22 individuals included in this survey showed any grave effects from their particular field of work. The previously cited report in 1911, by von Jagie, *et al.*, of the occurrence of lymphatic leukemia in four x-ray workers, if not interpreted as a coincidence, stands, most probably, as the

TABLE II

	Date	Hemoglobin	R.B.C.	W.B.C.	Polys.	Lymphocytes	Trans.	Eosinophiles
M. D.	5/35	88%	4,430	7,300	65	32	0	2
	8/35	80%	4,350	5,750	64	36		
	9/35	76%	4,510	6,580	67	32		1
	10/35	85%	5,040	7,600	63	37		
	10/22/35	85%	4,990	7,200	67	26	5	2
P. K.	1/35	82%	4,380	8,200	63	34	0	3
	3/35	80%	4,230	11,250	66	30		4
	4/35	77%	4,690	7,500	57	40		3
	8/35	81%	4,400	7,350	58	41		1
	10/9/35	90%	5,203	7,600	73	27		
	10/25/35	95%	5,922	6,200	65	33		1
E. V.	5/35	92%	4,390	6,800	53	45	0	2
	8/35	80%	4,300	8,750	52	43	0	5
	9/35	83%	4,580	7,000	67	29	0	4
	10/35	80%	4,920	11,000	66	34		
J. A.	1/35	80%	3,970	6,900	50	47	0	3
	3/35	80%	3,400	5,300	42	58		
	4/35	77%	4,660	6,200	66	33	0	1

supreme warning of the results of improper protection.

Our analysis rather definitely indicates that the hemoglobin and red cell counts are not affected. The one nurse who manifested an anemia probably had the dyscrasia long before she commenced radium work. If this assumption is not correct, then the action of ultra-hard gamma rays is different from that of x-rays. Experimental work by Mottram (8) demonstrated that the bone marrow is less affected by x-rays than are the lymphoid tissues, and it may be that the contrary holds true with radium rays. Nevertheless, this review conclusively points to the greater sensitivity of the white cell constituents of the blood.

The most constant and, evidently, the most characteristic alteration that occurs in the peripheral blood after exposure to stray radiation from either radium or x-rays is a decrease in the polymorphonuclears and an increase in the lymphocytes. Since a definite reduction in the total white cells was recorded in but two individuals, and an eosinophilia occurred in but two others; we cannot cite that as a definite blood change. This negation is further substantiated by the far greater percentage of white cell change which appeared in 20 individuals of the 22 studied. (See Table III.)

Whether we are justified in laying emphasis on the blood count reading as an index of reaction is a moot question. In 1921, Larkin (5) reported a case of acute aplastic anemia, which developed in an x-ray worker upon whom a blood analysis had been found to be normal just three

TABLE III

Group	Sex	Name	Years at Work	Hgb.	R.B.C.	W.B.C.	Change in P-L	Eos.
I	M.	A. K.	6	0	0	0	1+	0
	M.	T. W.	3	0	0	0	0	0
X-ray diagnosis	F.	W. F.	4	0	0	0	4+	0
	M.	L. F.	10	0	0	0	0	0
II	M.	G. K.	21	0	0	0−	7+	0
	M.	G. L.	7	0	0	0	4+	0
Radium element								
III (*a*)	M.	F. S.	3	0	0	0	1+	0
	M.	F. H.	6	0	0	0−	5+	0
	M.	G. C.	13	0	0	−	5+	0
Radium emanation: hospital								
III (*b*)	M.	A. K.	6	0	0	0	27+	+
	M.	S. S.	6	0	0	0	12+	0
	M.	G. E.	6	0	0	0	9+	0
	M.	R. L.	8	0	0	0	7+	0
	M.	W. B.	3	0	0	0	1+	0
Radium emanation: commercial								
IV	F.	M. M.	3	0	0	0	1+	0
	F.	V. J.	2	0	0	0	1+	0
	F.	D. I.	4	0	0	−	8+	0
	F.	C. S.	3	+	0	0	7+	+
X-ray therapy and radium								
V	F.	P. K.	1	0	0	0	3+	0
	F.	J. A.	6 mo.	0	−	0	3+	0
Radium pack nurses	F.	E. V.	6 mo.	0	0	0	2+	0
	F.	M. D.	6 mo.	0	0	0	1+	0
22 cases: 9 females; 13 males				1	1	4	20	2

+: an increase when referring to hemoglobin and eosinophiles; a change in the ratio with a decrease in polymorphonuclears and increase in lymphocytes.
−: a decrease.
0: no change.

months prior to the sudden onset of the disease. Will blood counts taken at varying or even regular intervals give adequate warning of an approaching danger?

It is not our purpose to undertake a discussion of the signs and symptoms evoked in these workers with the idea of establishing some specific sequence of symptoms characteristic of adverse conditions, but the symptom of undue tiredness which was dominant and often concomitant with alteration in the P-L ratio was persistently noticeable. These workers took heed of this change, emphasized greater precautions in the course of their work, enjoyed more sunshine and fresh air, and were often rewarded by a dissipation of the fatigue, and a more normal blood count level.

It appears, therefore, that until some more sensitive index is made available, close scrutiny of the ratio between the polymorphonuclears and lymphocytes should be made at regular intervals. When fatigue accompanies an altered ratio in these components, it will be well to emphasize precautionary and recovery measures.

CONCLUSIONS

(1) Individual sensitivity to stray ray exposure exists among radium and x-ray workers.

(2) A decrease in the polymorphonuclears and an increase in the lymphocytes is the one definite evidence of blood reaction to the stray rays.

(3) The hemoglobin and red cell levels are not altered.

(4) The stray rays from the x-ray diagnostic machines are least likely to produce any blood reactions.

(5) Greater emphasis on precautions provided in the laboratory, or a vacation from the routine, are usually adequate measures in returning the blood to more normal levels.

(6) Many workers will soon establish a different level in one or all of the white cell components which will constitute the normal for the individual.

55 East 86th St.

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INFLUENCE OF ROENTGEN RAYS AND RADIUM ON EPIPHYSEAL GROWTH OF LONG BONES[1]

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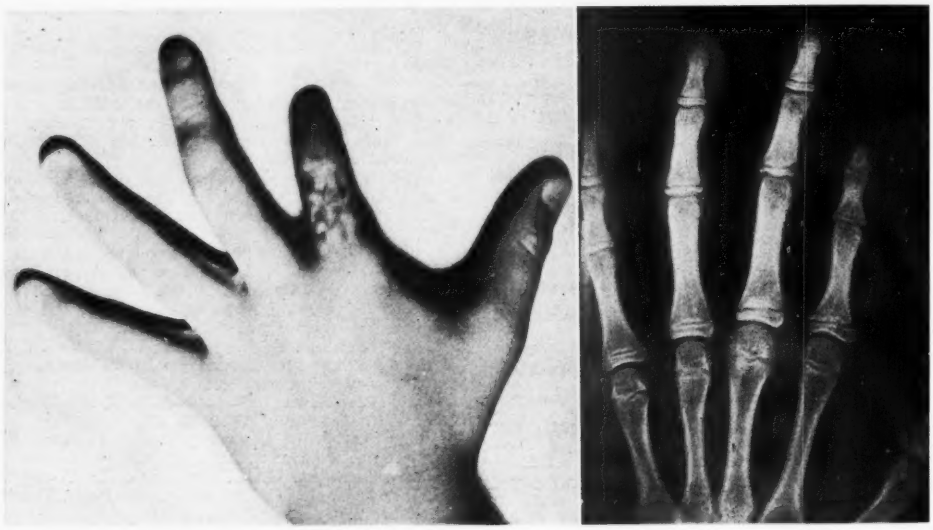
AMONG the unfortunate late results of x-ray and radium therapy are the deformities which result from disturbances in the longitudinal growth of irradiated long bones of children. This effect of roentgen rays was demonstrated experimentally by Perthes (1) soon after these rays had been discovered, and since then there have accumulated several reports both of illustrative clinical cases and of comprehensive experimental investigations. The most complete and recent study was reported by Brooks and Hillstrom (2), in 1933.

Although many observations herein recorded merely repeat and confirm those previously reported, the clinical importance of the subject and the apparent general lack of appreciation of its importance justify emphasis by repetition.

CASE REPORTS

Case 1. A. J., a girl five years of age, was seen in consultation by Dr. Hunt. Her parents were perturbed regarding the failure of her right index finger to grow. Two years previously, radium had been applied by another physician over a nevus on the dorsum of the finger, with no record of dosage. The nevus disappeared, but atrophy and telangiectasia gradually appeared. The finger failed to lengthen, eventually being shorter than the little one.



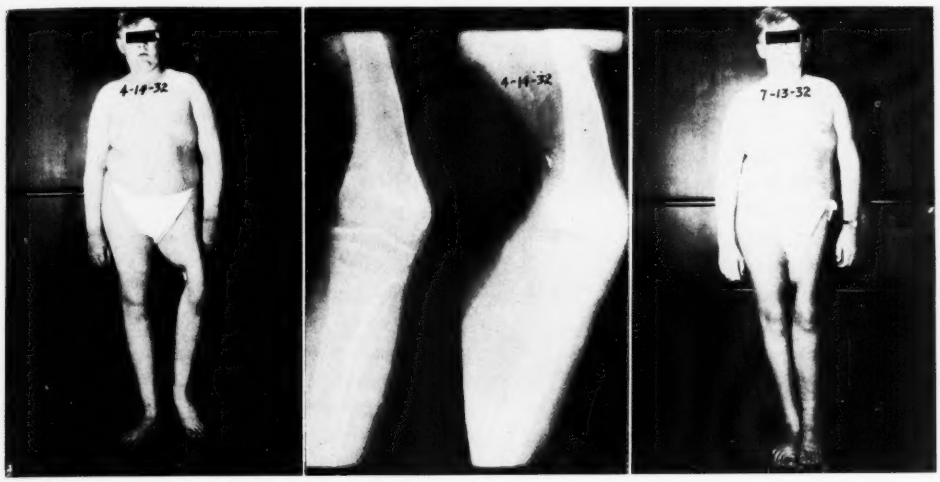
A *B*

Fig. 1-*A*. Hand of a child five years old. The index finger had been treated two years previously with radium. Note atrophy and telangiectasia on its dorsal surface, also that the index finger is shorter than the little one.

Fig. 1-*B*. The roentgenogram shows that all of the epiphyseal lines except the proximal line of the proximal phalanx have closed.

As illustrated in Figure 1-*A*, the right index finger was much shortened, and the

[1] Presented before the Radiological Society of North America, at the Twenty-first Annual Meeting, in Detroit, Dec. 2–6, 1935.



A *B* *C*

Fig. 2-*A*. A boy of 17 years, with shortening and genu varum of the left leg, resulting from irradiation with x-ray and radium over the mesial aspect of the lower end of the femur, which he received 12 years previously.

Fig. 2-*B*. The roentgenogram shows varus distortion of the lower end of the femur. The epiphyseal line on the mesial aspect is closed, with fusion of the shaft to the epiphysis at this area, preventing growth. The line is open laterally, allowing continued growth there which has adducted the lower end of the femur.

Fig. 2-*C*. The same boy three months later, showing correction of the deformity and of some of the shortening by means of a supracondylar osteotomy.

skin presented the atrophic and vascular changes which result from intensive irradiation, relatively rich in beta rays. The roentgenogram, Figure 1-*B*, shows that all epiphyseal lines of this finger except the proximal one of the proximal phalanx have closed. Thus, growth from all but one source has been irrevocably terminated.

Case 2. Q. M., a boy 17 years of age, presented a genu varum deformity, with 2.5 inches shortening of the left leg. He gave a history that 12 years previously a tumor on the mesial aspect of the knee had been irradiated with both radium and x-rays. There developed a varus deformity and shortening, which progressed until two years ago (Figs. 2-*A* and 2-*B*), when the bowing and some of the shortening were corrected by a supracondylar osteotomy of the femur (Fig. 2-*C*). Since then some of the bowing has recurred. In addition to the slight deformity and a shortening of 2.5 inches, there is a large, soft tissue defect covered by a thin telangiectatic scar.

Roentgenograms taken two years ago show that the epiphyseal line had closed on the mesial aspect but was still present on the lateral side (Fig. 2-*B*). Growth, continuing on the lateral side but not on the mesial side, had thus twisted the lower end of the femur into a varus deformity.

EXPERIMENTAL STUDIES

Five litters of rabbits from three to five weeks old were used. In each animal one extremity was irradiated and the opposite one served as a control. All of the body except the area to be treated was protected from the rays. Growth was measured every three weeks by roentgenograms, taken with the leg in direct contact with the x-ray film and with a constant technic. Final comparative measurements were made from the gross specimens obtained at necropsy. Optimum circumstances for growth were maintained, all animals remaining with their mothers during the full period of lactation.

SERIES I. EXPERIMENT

Growth Uninfluenced by Irradiation of the Shaft Only.—Since all growth in the length

of long bones takes place at the ends of the shafts, and is dependent upon the osteogenic activity of the cells of the epiphyseal cartilaginous discs and of the zones in the diaphyses immediately adjacent to the discs, growth disturbances should not occur so long as these cells are not included in the area to be irradiated unless they receive sufficient secondary irradiation from a spread within the shaft to cause cell damage. To determine this point, the left femoral shafts of three rabbits three weeks old were given 1,540 r measured in air over 10 cm. field with 5 milliamperes, 130 kilovolt peak at 30 cm., through $^1/_4$ mm. of aluminum, effective wave length about 0.50 Ångstrom. Allowing 350 to 400 r as the erythema dose (3), this is about four erythemas. The ends of the shaft and the epiphyseal cartilages were protected from the primary rays by 2 mm. of lead.

After two weeks, most of the hair over the area on the side exposed to the rays (the lateral side) came out, but later was partially replaced. One rabbit was killed 14 days and two were killed 90 days after treatment. From serial roentgenograms and from measurements of necropsy specimens, there was no evidence that normal growth had been disturbed at any time. Histologically, the epiphyseal cartilages and the adjacent new-formed bone were normal.

SERIES II. EXPERIMENT A

Minimum X-ray Dosage Required to Provoke Growth Retardation.—The entire left legs of three rabbits, four weeks of age, were irradiated with doses of approximately 100, 80, and 50 per cent of that required to produce erythema in man. They were killed 167, 172, and 162 days later, respectively. The dosage and technic were as follows: rabbits (*B*, 8) 400 r; (*B*, 12) 300 r; (*B*, 7) 200 r, energized by a current of 5 milliamperes, 130 kilovolt peak filtered through $^1/_4$ mm. aluminum, given at 30 cm. distance.

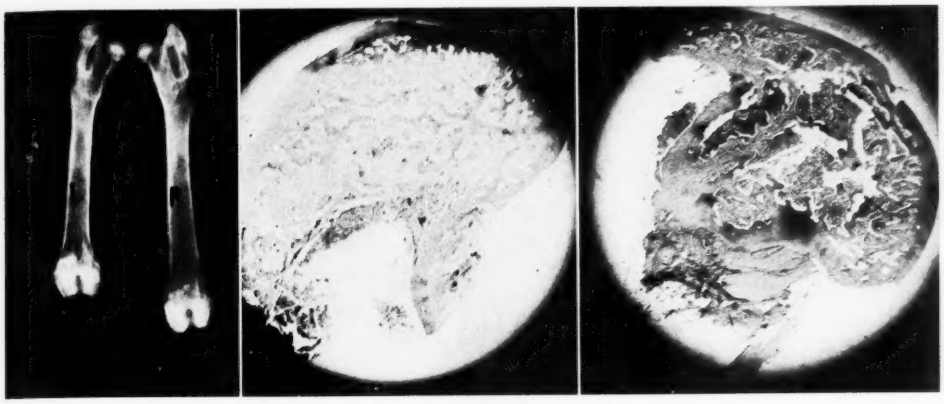
In the two rabbits which received 300 and 200 r in air, there was at no time evidence of a disturbance of growth in either serial roentgenograms or in the gross specimens. All bones of the irradiated and control extremities were equal in length. Histologically, however, the treated epiphyseal cartilages showed distinct evidences of damage. In cross-section, the discs varied greatly in thickness, in some areas separating the epiphysis and diaphysis by a very thin layer. The cells were arranged somewhat irregularly, preserving only partially the normal pattern of regular columns.

The femur of the rabbit which received the larger dose (400 r in air) was 5 mm. shorter than the control. Microscopic sections through both ends of the bone revealed great irregularity in the thickness of the epiphyseal cartilaginous discs, and some abnormal inconstancy in the size and arrangement of the adjacent trabeculæ of new bone. The cartilage cells varied in size abnormally. In some areas they were arranged in fairly regular columns, and in others there was little or no semblance to the normal; the cells being scattered and showing no grouping into columns whatsoever.

There was definitely less hemapoietic tissue in the marrow of the epiphyses and of the ends of the diaphyses than normal. None of the animals developed either a definite erythema or a loss of hair. Thus, exposure of the epiphyseal cartilages to roentgen irradiation, somewhat less than that dosage required to induce an epilation, caused retardation of growth. Although lesser doses did not influence the rate of growth, they did inflict obvious damage to the cartilage. To determine the minimum amount of irradiation necessary to cause histologic changes in the cartilage, the second section of this series of experiments was carried out.

EXPERIMENT B

Minimum Dosage Causing Histologic Change in the Cartilage.—In each of three rabbits, four weeks old, the entire left leg was irradiated, and the animals killed 14, 15, and 12 days later. The factors used were 5 ma., 130 kv., 30 cm. distance, and



A *B* *C*

Fig. 3-*A*. Both femurs of Rabbit *B-19*, showing shortening of 15 mm. in the left one as a result of irradiation with 1,540 r 191 days previously.

Fig. 3-*B* and 3-*C*. Photomicrographs of longitudinal sections through the condyles of these femurs show that the epiphyseal cartilage has been completely ossified and obliterated in *B*, the irradiated femur, but is still present in *C*, the normal one.

1/4 mm. aluminum filtration. The doses and results follow.

(1) *Rabbit B-21* received 300 r, approximately 80 per cent of an erythema dose, the histologic findings 14 days after irradiation being as follows: The cartilaginous discs were wider than those of the normal control. The cells in some areas were arranged in columns, which were fairly regular but distorted, and in others they were scattered and so disorganized that there was no suggestion of columnar arrangement. The cells varied greatly in size, some being abnormally large. In many cells the nuclei stained poorly and appeared to be shrunken. In areas in which the columns had been partially retained, there was an abnormally large number of empty lacunæ. The intercellular matrix appeared to be diminished. There was an astonishingly large reduction in the amount of hemapoietic marrow.

(2) *Rabbit B-22* received 200 r, approximately 50 per cent of an erythema dose. Fifteen days later there were essentially the same changes as described above, with the exception that the columnar formation had been lost nowhere although it was distinctly distorted. The hemapoietic tissue was diminished in amount as compared to the control.

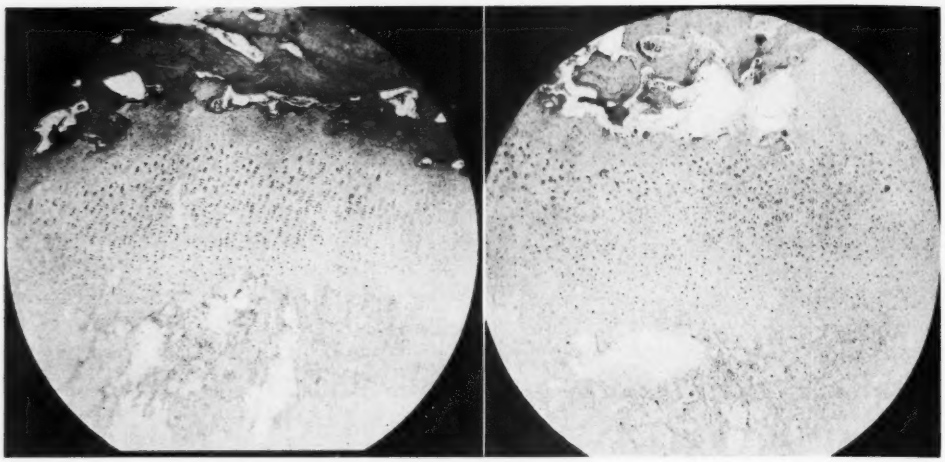
(3) *Rabbit B-23* received 100 r, approximately 25 per cent of an erythema dose. No abnormality in the cartilage could be detected. The marrow, however, contained less hemapoietic tissue than normal.

COMMENT

X-ray dosage of about one-half of an erythema dose produced distinct evidence of injury to the cells of the epiphyseal cartilage, and a dosage of about one-fourth caused no evidence of injury to cartilage, but did cause insult to the hemapoietic tissue. Thus, cartilage is slightly less radiosensitive than hemapoietic tissue, and its threshold in the rabbit lies somewhere between one-half and one-fourth of an erythema dose.

SERIES III

Histologic Changes at Various Time Intervals after Irradiation.—The progressive changes which take place in cartilage after injury from a large dose of x-rays were observed in seven rabbits, three and four weeks old. The experiment was designed



A *B*

Fig. 4 (*A* and *B*). Low power photomicrographs of longitudinal sections of the distal epiphyseal lines of both femurs of Rabbit *B-10* at age of five weeks. (*A*) Cartilage of the normal femur. (*B*) Cartilage of the irradiated femur, six days after exposure to 1,540 r of x-ray. Note increased thickness of cartilage and loss of column formation of cells.

particularly to determine the extent to which cartilage recovers from injury and the readjustment which takes place with recovery. The entire left leg of each animal was irradiated with 1,540 r, given with 5 ma., 130 kv. at 30 cm. distance, and through $^1/_4$ mm. of aluminum.

The animals were killed at periods varying from two to 191 days after irradiation, and the following observations made of the microscopic sections:

(1) *Rabbit B-9.*—The cartilage two days after irradiation shows extensive disorganization. The cells have moved out of line and the columnar pattern has been largely lost. Many cells are larger than normal and the nuclei of many stain poorly. The matrix appears to be diminished in amount. A photomicrograph appears in Figure 5.

(2) *Rabbit B-10.*—Sections six days after irradiation (Fig. 4) show complete loss of the normal columnar pattern of cells. Many are abnormally large and others small, the size varying greatly. The nuclei of many stain poorly and are shrunken. The matrix appears to be diminished in amount. Nearly all of the hemapoietic tissue of the marrow has disappeared.

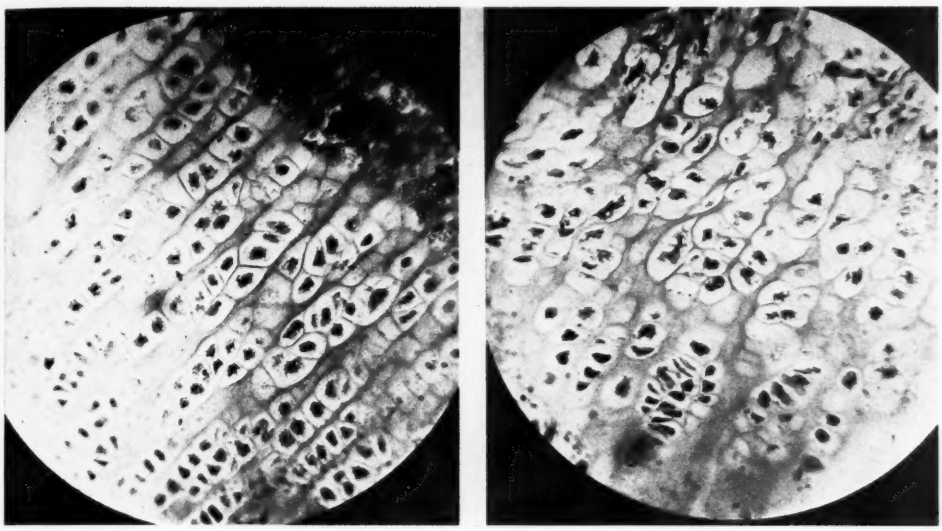
The cells in the deep layer of the articular cartilage show the same changes. There was no shortening of the bones of the irradiated leg.

(3) *Rabbit B-11.*—Fourteen days after irradiation, the picture is essentially the same as that of Rabbit *B-10*, except that the new-formed trabeculæ of bone appear thicker and more irregularly disposed, and, definitely, there is present more hemapoietic tissue.

The femur of the irradiated leg was 4 mm. shorter than the normal one.

(4) *Rabbit B-16.*—After 36 days, the columnar pattern of cells has reappeared in many areas but the rows are imperfectly formed, being short and irregular. The cells are more constant in size and the nuclei stain well. Many lacunæ appear to be abnormally empty. The most striking change is the development of great variation in the thickness of the cartilaginous disc, which is quite narrow in certain areas. (See Fig. 6.) Hemapoietic tissue is fairly abundant, but there appears to be less than the normal amount.

The femur of the irradiated leg was 10 mm. shorter than the normal one.



A *B*

Fig. 5 (*A* and *B*). High power photomicrographs of longitudinal sections of the distal epiphyseal lines of both femurs of Rabbit *B-9*, at age of 30 days. (*A*) Cartilage cells of normal control femur. (*B*) Cartilage cells of irradiated femur, two days after exposure to 1,540 r. Many cells are abnormally large and swollen and have moved out of line, thus disorganizing the normal arrangement of columns. Many nuclei appear distorted and some take the stain poorly. The matrix appears to be diminished.

(5) *Rabbit B-17.*—The changes after 98 days differ from those of Rabbit *B-16* in the greater variance in the thickness of the cartilaginous disc. In some areas the epiphysis and the diaphysis are almost in contact, and in others they are separated by a layer of cartilage of nearly normal thickness. In the widest areas there is little or no row formation.

The irradiated femur was 13 mm. shorter than the normal one.

(6) *Rabbit B-18.*—In the 191-day specimens, the irradiated epiphyseal cartilages have become completely ossified. No cartilage was seen. In the control femur there was still present a thin layer of cartilage of irregular thickness. The cells were few in number and scattered without pattern.

The irradiated femur was 18 mm. shorter than the normal one.

(7) *Rabbit B-19.*—The results were the same as those in Rabbit *B-18*, except that the epiphyseal cartilage at the distal end of the control femur was thicker and contained more cells, and these were arranged in very short columns.

The irradiated femur was 15 mm. shorter than the normal one. (See Fig. 3.)

Serial roentgenograms were made of the four rabbits which survived longer than four weeks after irradiation. Measurements from these showed that growth continued after irradiation, but at a slower rate, that is, it was merely retarded and not arrested. This retarded rate remained constant until normal cessation of growth was nearly reached. Growth then diminished abruptly and stopped prematurely. In each of the same four rabbits there developed loss of hair and an intense erythema on the lateral side.

SERIES IV

Retardation of growth becomes less as dosage is divided and as interval between divided doses is increased.

The entire left leg of five rabbits four weeks old was irradiated with 1,260 r, administered with 5 ma., 130 kv. p. at 30 cm. distance, and with $^1/_4$ mm. aluminum filtration. All animals ultimately received

Fig. 6. Low power photomicrograph of a longitudinal section of the distal epiphyseal line of the left femur of Rabbit *B-16*, at the age of 59 days, and at 36 days after irradiation with 1,540 r. Note scattering of cartilage cells, but with partial restoration of the normal alignment of cells into columns. Note also the irregular thickness of the cartilage; in many areas it is very thin.

the same total dosage but received it in divided doses, which were varied as to number and as to the time interval which elapsed between the divided exposures.

All were killed 120 days after the initial treatment, the bones measured, and histologic studies made of the cartilages. These studies showed essentially the same changes in the cartilages as described in Series III, Rabbit *B-17*.

The results, the disturbances to growth, and the reactions of the skin are tabulated in Chart I under section Series IV. The extremities of the rabbits which received the total dosage in two treatments developed more shortening than did those of the litter mates receiving the same dosage in three treatments. Two treatments given two days apart caused greater shortening than did two treatments of the same total dosage administered seven days apart. Three treatments at two-day intervals produced more shortening than did the same treatments at four-day intervals, and the latter more than the same treatment at seven-day intervals. In general, three treatments of 400 r at weekly intervals gave about the same effect as the single dose of 400 r.

As in the rabbits of Series III, serial roentgenograms showed that growth in the irradiated leg had never ceased, but had continued at a retarded rate which remained constant until the animals were killed.

DISCUSSION AND CONCLUSIONS

(1) Two clinical examples of deformity from retardation of longitudinal growth of bone by intensive irradiation are presented.

(2) The shaft of a bone can be irradiated with 1,500 r at 0.50 Ångstrom unit, without causing a disturbance of growth providing the epiphyseal cartilages are protected from direct action of the rays. The intensity of secondary irradiation generated produces no demonstrable insult to the cartilage.

(3) In young rabbits, a single exposure of 400 r or a dose about equal to that required to produce an erythema in man caused a slight retardation of epiphyseal growth in long bones. Dosages less than half of that amount produced histologic evidence of injury to the cells of the epiphyseal cartilages.

These results, of course, are not directly applicable to human bones, in which there is a very much thicker layer of soft tissues, cartilage, and bone to be penetrated. The growth in the rabbit is much more rapid and its span very short. Also, it is possible that cartilage is more radiosensitive in the rabbit than in man.

However, it may be deduced that, since a dose sufficient to retard growth in the rabbit did not produce a definite erythema, the epiphyseal cartilage is more radiosensitive than skin, and that, by the same token, an erythema dose over an epiphysis in a child carries with it the danger of retardation of growth.

(4) Fractionation of the total dosage of x-rays, and protraction of the interval between the fractionated units, greatly lessens the effect upon growth. This well-known principle should be applied, if possible, in administering therapy to the region of an epiphyseal line in children.

(5) All evidence indicated that the action of x-rays upon cartilage cells is solely

destructive. Simon (4) has reported that repeated small doses of roentgen rays stimulate osteogenesis and increase callus formation in experimental fractures.

(6) The histopathology of the cartilage and marrow resulting from irradiation is reported. Definite and extensive changes in the cells and architecture of the cartilage were present as early as two days after exposure. The appearance of explosive disruption of the normal columnar arrangement of cells is apparently partially permanent, but in some areas the normal architecture becomes imperfectly restored. With recovery of surviving cells, there develops great irregularity in the thickness of the layer of cartilage. Even after large doses, growth continued but at a slower rate than normal. Growth also terminated prematurely, the epiphyseal line closing

CHART I.—TABULATION OF EXPERIMENTS

Exp.	Rabbits	Age in Weeks	Total Roentgen Units	No. of Exposures	Interval between Exposures	Loss of Hair: Erythema	Shortening of Femur by Mm.	Days after Irradiation	Histology of Epiphyseal Cartilage
I	B-13, B-14, B-15	5	1,540	one		yes	none	90	Normal
II	B-8	4	400	one		no	5	167	Irregularity of thickness of epiphyseal cartilage and of rows of cartilage cells
	B-12	5	300	one		no	none	172	As in B-8, but probably of lesser degree
	B-21	4	300	one		no	none	14	Irregular thickness of disc and of rows of cartilage cells: cell damage (see text)
	B-7	4	200	one		no	none	162	Some variance in thickness of epiphyseal cartilage. Cell columns disorganized
	B-22	4	200	one		no	none	15	Column arrangement of cartilage cell disorganized. Large swollen cells
	B-23	4	100	one		no	none	12	Normal
III	B-9	4	1,540	one		no	none	2	Disorganization of columns of cells: cell damage (see text)
	B-10	4	1,540	one		no	none	6	As in B-9
	B-11	4	1,540	one		no	4	14	As in B-9 (see text)
	B-16	3	1,540	one		yes	10	36	Less disorganization of columns. Greater irregularity in thickness of disc
	B-17	3	1,540	one		yes	13	98	Disorganization of cell columns. Greater irregularity in thickness of disc
	B-18	3	1,540	one		yes	18	191	Epiphyseal lines closed but still open in control
	B-19	3	1,540	one		yes	15	191	As in B-18
IV	B-1	4	1,260	two	2 days	yes	14	120	As in B-17 (see text)
	B-2	4	1,260	two	7 days	yes	10	120	As in B-17
	B-4	4	1,260	three	2 days	yes	10	120	As in B-17
	B-5	4	1,260	three	4 days	no	8	120	As in B-17
	B-6	4	1,260	three	7 days	no	5	120	As in B-17

earlier than the normal. In Case 1, a presumably intensive dose of radium caused complete destruction of the epiphyseal cartilages, with prompt ossification and immediate growth arrest.

(7) It is obvious that individual cartilage cells vary considerably in respect to radiosensitivity. Some appeared severely damaged and others quite normal. In explanation, Brooks and Hillstrom (2) offer a logical hypothesis. They believe that cartilage cells normally vary in respect to duration of life and capacity for cell division, and that those endowed with a shorter life span and with greater capacity for rapid cell division are more susceptible to irradiation, many of them being destroyed. Thus they explain the fact that the period of growth is not appreciably shortened, although the rate is retarded.

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DISCUSSION

DR. EDWIN C. ERNST (St. Louis): I have felt for some time that we were paying a great deal of attention to cancer conditions—malignancy—but perhaps forgetting the wonderful possibilities of the use of the x-ray in proper doses in non-malignant conditions, inflammatory conditions, and other diseases. I think the field is unlimited. In some instances we have been a little afraid to go ahead and use the x-ray in other conditions beside malignancies.

I am sure Dr. Kelly does not wish to leave the impression that the use of portable machines in treatment work, except to emphasize that the amount of radiation is relatively small, is desirable. Sometimes a paper of that sort is misunderstood.

DR. PAUL MOORE (Muncie, Indiana): I have a case report to present which I have discussed with Dr. Pfahler and which he thought was worth while bringing before the Society.

I do not know how many of you have treated cases of hemolytic streptococcus septicemia with a cure, but in view of the fact that this is a decidedly fatal disease, any simple measure such as roentgen therapy which results in cure certainly is a worth-while procedure.

This patient was an adult male, about forty years of age, whom I knew as a healthy man. He had a bilateral acute mastoiditis, with a bilateral mastoidectomy. After he left the hospital he developed the complication of a lateral sinus thrombosis. He returned to the hospital for operation and a clot was removed from the lateral sinus. This resulted in further complications, namely, a hemolytic streptococcus septicemia.

His condition became worse. Each complication was worse and more grave. He was transfused frequently. He would recover slightly but his condition became progressively worse. Daily his blood culture was positive for hemolytic streptococcus; the white count was very high: his temperature was 104 degrees.

Dr. Gatch, Professor of Surgery at the University of Indiana, was called in consultation and reviewed the case. His opinion was that everything had been done that could be done. However, in view of the fact that several cases of streptococcus endocarditis had recovered following roentgen therapy at Indianapolis, he suggested the use of x-ray therapy in this case.

When I saw the man he was practically moribund; he was so weak he could not talk although I think he was conscious. His anemia was apparent from his lemon yellow skin, and one's reaction to the case was that the next zephyr of air or change of temperature might cause him to die. His condition was exceedingly grave, so grave that I hesitated to treat him and take the blame for his death.

However, without any particular knowledge of dosage, I selected 50 roentgens anterior to the spleen and 50 roentgens anterior to the liver, with the idea of treating the reticulo-endothelial system on the theory not only of stimulation, but rather of destruction of reticulo-endothelial cells and on the theory of the release of bactericidal content of these cells.

The following day the patient spoke. This, however, did not influence us except that we thought we would treat him again. We treated him the second time and the following day he said, "This is the first good night's sleep I have had for weeks." That encouraged us.

At the end of four days he was sitting up in bed, his blood stream was pure, and negative for hemolytic streptococci. His temperature was down to normal and his blood count was almost normal. The response was so dramatic that it certainly left a great impression upon me.

It is true that cases of this disease can recover rapidly, once they establish immunity in some way of a crisis, as we see in pneumonia. However, this man's condition had become progressively worse in the face of successive complications, each one of which was certainly more grave than the one before it. At the end of the week this man had a dinner party in his room for a group of friends.

Incidentally, I failed to mention that he had developed deep abscesses interosseous between the radius and ulna, interosseous between the tibia and fibula, an abscess against the cervical spine, and sub-scapular abscesses, all of which were surgically drained. As to the presence of deep abscesses in liver, spleen, brain, kidney, etc., I do not know. It would seem almost the complication to be expected.

He made an uneventful recovery except for the development of an infectious arthritis in one shoulder joint, but this responded very nicely to simple physical therapy. This treatment was given three years ago and the patient is perfectly well and normal at this time.

In conclusion, I would say that certainly no claim should be made in a case such as this—that we should not definitely state that the patient was cured by means of roentgen therapy, but certainly it is worth further study and it certainly was a striking response.

I would mention, under the subject of the treatment of inflammatory conditions, that in a small series of cases I have had excellent results from the treatment of both acute and chronic mastoiditis. One child had roentgenograms of both mastoids, with a diagnosis of bilateral acute mastoiditis. I recommended surgical intervention because of the breaking down of the cell walls. The otolaryngologist insisted on surgical intervention, but the family doctor decided he wanted x-ray treatment (the child was a nephew of his). The response in this case was immediate and excellent.

I would just mention, also, that the x-ray treatment of tenosynovitis, both acute and chronic, has been very successful in my hands in a small group of cases.

DR. JAMES F. KELLY (Omaha, Nebraska): I wish to thank the Chairman for calling to my attention the fact that I did not mention any technic. In following the slides and not referring to my written text I overlooked it.

The technic we recommend at this time is as follows: On extremity work we use from 90 to 100 kv., about 100 r per dose; that is, 100 r in the morning and 100 r in the evening for three days. We used a 15-inch target-skin distance.

On the trunk cases we increase the penetration, using from 130 to 160 kv.; we also increase the filtration, using 3 mm. aluminum up to one-quarter mm. copper. The r per dose is the same and the time interval, a dose in the morning and a dose in the evening, is also the same. We treated one abdominal case which followed a gangrenous appendicitis with 160 kv., one-quarter mm. copper, and the response was very prompt.

There is another thing I forgot to mention—the effect of the x-ray therapy is evident in from twelve to twenty-four hours.

The clinical evidence of improvement is marked. In one case, when I was out of the city the Sister in charge of the X-ray Department insisted on treating an 8-year-old boy with x-ray in preference to amputation. The surgeon said "I will give you until sundown to show improvement; if the patient does not show improvement then, I must cut the arm off." The boy improved the first day and continued to improve and returned home well, retaining his arm. I wish to apologize for seeming critical of the surgeon, but anyone who has had an experience similar to mine would be somewhat of the same opinion, I am sure.

For instance, the third case we treated required amputation. This was all right; the surgeon amputated at the knee and serum was injected in the thigh. The disease—or the gas and discoloration at least—went right through the injected area and proceeded up to the insertion of the muscles about the pelvis. (This will probably answer Dr. Hunt's question in regard to amputating gangrenous areas.) It was black when we first started to treat it, yet all of this tissue recovered its normal color, so that all of the tissue this patient lost was that due to his shotgun injury and his first amputation. No higher amputation was required. However, the lower amputation was justified, the leg having been practically blown off just below the knee.

After this experience the same surgeon a short time later saw the case of an old man who had been hurt with a harrow—an ankle or foot was hurt. The surgeon did not consider x-ray treatment for the gas gangrene which developed, but promptly amputated near the ankle. He also used all the serum which he thought was necessary. The disease progressed up the leg in spite of the first amputation. He then amputated below the knee, but the disease showed evidence of going higher on the leg so he gave the x-ray department a chance. The patient responded promptly to x-ray treatment. Such experiences as these have built up an aggressive attitude, if not a critical one, on the part of the surgeon.

I think what I just said will tell Dr. Hunt about all we know in regard to the color of the tissues. We have had much of this discolored tissue recover. It is evidently not a true gangrene—probably more of a cyanosis.

One should certainly treat well beyond the lesion. We usually take multiple fields. We treat at a 15-inch distance if the entire limb and part of the trunk is to be covered. We also allow a certain amount of overlapping at the borders of the fields.

Possible effects of the x-ray were loosely covered with the first presentation of cases in 1931. I think the formation of hydrogen peroxide in the tissues is certainly something to think about, and I believe the men in California deserve credit for that observation. Dr. Emery promised he would give me some further information on this matter later. He was unable to stay for this symposium.

The important thing, I believe, is that the x-ray should be tried in all cases of gas gangrene, regardless of the color of the tissues or the condition of the patient, and that amputation, unless necessary for some other reason, is not indicated for the gas gangrene.

The following questions were answered as follows during the discussion:

1. Dangers?—In the average hands, since the course of the disease is very short, it seems that no danger of over-radiation is present.

2. Complications?—No complications due to radiation effects have been met.

3. Limitations?—It must be distinctly understood that the x-ray should not be used independent of the use of serum, especially anti-tetanic serum.

4. Frequency and Duration of Treatment?—The influence or importance of the time factor in the successful treatment of inflammatory processes, as it is more fully understood, will probably prove to be as important as we now think it is important in the treatment of neoplastic processes.

5. Reason for Beneficial Action of the X-rays?—The probable formation of H_2O_2

in the tissues is suggested but has not been definitely proven.

6. Dr. Hunt's question as to how to decide on what gangrenous tissue to excise or amputate and what to leave cannot be answered definitely, but much of this darkened, apparently dead tissue recovers fully after radiation and, as stated before, this color is probably due to a localized cyanosis and is not a true gangrene.

DR. HOWARD B. HUNT (Omaha, Nebraska): It appears to have been well established that an erythema dose for man of intermediate roentgen rays, applied to the skin over a rabbit's epiphysis, produces definite retardation of growth and subsequent asymmetry. It may be assumed that the great majority of this dosage was delivered into the tissues, considering that the epiphyseal cartilage is only about 3 mm. below the surface and the small bulk of the tissues offers only slight opportunity for generation of secondary radiation.

Radiography in children is usually conducted with about 5 roentgens, and not over 50 roentgens exposure over any one area. This dosage gives no evident microscopic changes in cartilage and is free from danger as clinical practice has well established. Fluoroscopy for the removal of foreign bodies is occasionally so prolonged by unthinking determination as to produce a dermatitis which, if given about the active epiphyseal zones in a child, carries the possibility of a subsequent gross asymmetry of growth. The extent of deformity varies with the rate of normal local growth. Fractures most commonly manipulated are about the elbow and in the shafts of the bones where little or no effect upon total growth will result. In general, no disturbances in growth are to be anticipated following radiographic or radioscopic study.

Radiotherapy in children, particularly of benign lesions, must at times be considered from the standpoint of possible subsequent retardation of growth and deformity. For example, in treating the thyroid of a child we shield the epiphyseal centers at the medial ends of the clavicles. A small nevus vasculosus about the wrist or knee is treated by coagulation or carbon dioxide ice rather than by radium, although radium is used over the shaft and less active epiphyses with relative impunity. Overtreatment of hemangiomas should be avoided by allowing at least three and preferably six months for evaluation between applications. Benign osteomas which are common about the knee should be left alone or resected and not irradiated, due to their radioresistant nature and subsequent bony deformity from disturbed growth. Relatively soft rays would appear best adapted to treatment of the skin in children without appreciable penetration of rays to the growing epiphyseal tissues. The treatment of proven malignancies justifies a radical attack which ignores the possibilities of any subsequent deformities.

The application of roentgen rays to the therapeutic control of bone growth is limited to few conditions. We have recently treated a local gigantism of two toes in an infant apparently due to increased blood supply secondary to an adjacent plantar cavernous hemangioma, 500 roentgens in air through 2 mm. Al were given over the toes and 400 mg.-hr. over the nevus in this case. Treatment is too recent to allow evaluation. Inequalities of bone growth are also encountered following infection or trauma, and from congenital or unknown causes. Although radiation is unable to accelerate growth in a more slowly growing zone, it can retard growth in the more rapidly growing opposite side. The relatively more rapid growth may be the result of stimulation by disease or merely a state of normal growth. It is essential that the treated zone should be homogeneously irradiated. Variable intensities of irradiation would lead to unequal retardation of growth and finally to a bowed distorted bone.

DR. FRICKE (closing): As to Dr. Hunt's two questions, first about the fractional treatment—the larger lesions we treat in two or three applications. We do not cover the same area twice except in the 20 per cent

of our cases, in which the condition has not subsided after a two- or four-day interval. We re-treated those.

About parotitis from irradiation, we have seen no cases following our technic. This appears to be an entirely different form of parotitis, on a mechanical rather than an infectious basis. In Baltimore, at the Howard A. Kelly Hospital, where we used the large radium pack to treat tonsil infections and to treat malignancies around the angle of the jaw, "irradiation" parotitis was an almost daily occurrence. We often had to warn patients that they would have this condition following a massive dose, but the condition always subsided in twenty-four hours with no treatment whatever and there was no discomfort from it.

SOME LAWSUITS I HAVE MET AND SOME OF THE LESSONS TO BE LEARNED FROM THEM[1]

(*Fifth Installment*)

By I. S. TROSTLER, M.D., F.A.C.R., F.A.C.P., *Chicago*

EXPERT TESTIMONY BY DEPOSITION

IN quite a number of malpractice and personal injury suits, the writer has given testimony by affidavit or deposition, for use in the trial of these cases elsewhere, mostly at a considerable distance.

In 1925, by stipulation between the attorneys acting for both sides of a medical malpractice suit resulting from x-ray treatment of eczema (in which $25,000 damages was claimed), in a southwestern State, certain questions were prepared and submitted to me for answers, the same to be introduced as evidence at the time of the trial. These questions were as follows:

"*Direct Interrogatory No. 1:* Please state your name, age, place of residence, and occupation."

Answer: . . .

"*Dir. Int. No. 2:* State what experience you have had with the use of the x-ray machine."

"*A.* I have been using x-ray machines and apparatus since 1896. Have specialized in x-ray work since 1907."

"*Dir. Int. No. 3:* State what types of x-ray machines and tubes you have used."

"*A.* I have used static machines, Ruhmkorff coils, Tesla coils, open and closed core transformers, the gas tubes and Coolidge tubes: I believe, all the different types of apparatus used in x-ray work in this country."

"*Dir. Int. No. 4:* State whether or not, in your opinion, and from your experience in x-ray work, a treatment with Victor Wantz x-ray machine, standard medium focus vacuum Coolidge tube, time of exposure five minutes, spark gap three inches, milliamperage five, distance 14 inches, no filter, would be considered a safe dosage in treatment of eczema."

"*A.* In my opinion, that would be a very safe dosage. It is about three-eighths of an erythema dose, or, to be more precise, it is exactly 75/196ths of the amount of x-ray which would produce a reddening of the skin comparable to a sunburn on an average individual with normal ordinary skin and the usual tolerance to the x-ray. Two and one-half times the dosage described in the question would still be within the amount of dosage considered as a safe single dose."

Cross-interrogatories

"*Q. 1:* If a patient having a small rough place on his arm indicating eczema or ring worm is treated by an x-ray, where after the x-ray is applied the affected part within a week appears to be burned, and for which burn the operators of said x-ray treat the patient for approximately ten months, the arm becomes swollen to treble its natural size, that no improvement in the condition of the arm appears, but the condition becomes so serious that an operation was necessary and was made by Dr. N. M. P., of X . . . . . Street, X . . . . . ., which operation consisted of removing a portion of the flesh of arm, approximately seven inches long and five inches wide, scraping of the bone and the affected part covered by a skin-grafting operation, then would you say that the x-ray was a safe dosage in the treatment and that the machine was in proper condition and that the operators properly and carefully applied same?"

"*A.* Because of the involved character of the question and the statements made therein, I am unable to answer any more than to say that the condition described in the question might be the result of proper x-ray treatment with safe dosage and the

[1] The Editor desires to state that much of the matter herein has to be printed in form as received, without normal punctuation or phrasing.

machine operating properly and the operators properly and carefully applying same, because other elements such as the presence in the patient of a marked idiosyncrasy or hypersensitiveness of the skin or other tissues of the patient to the x-rays, or a considerable increase in the voltage of the electric current in the service line which supplies the electricity to the x-ray machine, or that there was applied at some time shortly before or shortly after the x-ray treatment, some irritating or escharotic drug, lotion, salve, ointment, powder, or some other medication to the area treated, which would enhance the effect of the x-rays upon the tissues so applied, and to a great extent increase the likelihood of a burn following x-ray treatment, or the combination of any two or of all three of these factors. In the event of these factors or causes being present and operative, there would be more likely to be a burn and the machine still be operated properly, the dosage safe, and the treatment properly and carefully applied."

"*Q. 2:* Is it not a fact that if an x-ray machine is in good condition, properly adjusted, placed the proper distance from the point to be treated and left for the proper length of time, that it will not burn the part nor produce any injurious effects?"

"*A.* There are so many elements which govern x-ray treatment that this question cannot be answered without explanation. Even if the x-ray machine is in good condition, properly adjusted, the x-ray tube placed at the proper distance from the area to be treated, and the exposure made for the proper length of time, yet burns have occurred and do occur because of what is called idiosyncrasy or of hypersensitiveness of the tissues of the patient to the x-rays, and there is no way by which the presence of this idiosyncrasy or hypersensitiveness can be diagnosed or determined beforehand. It is considered the rule among x-ray therapists that the patients must assume this risk, if they want the x-rays applied."

The jury gave the plaintiff a judgment of $5,000: he settled for something less.

A LOUISIANA CASE

Mainly as an aftermath and result of the physician starting suit for his fee, the husband of a deceased patient started a suit for malpractice against a physician and his deceased partner's estate, alleging that the demise of plaintiff's wife was due to injuries resulting from a dermatitis or burn produced by x-rays applied by the deceased partner of one of the defendants, and suing for damages in the amount of $78,792.

The writer testified by affidavit, the testimony being much similar to that in the preceding case.

The trial was before a judge only, and decision was in favor of the plaintiff and against the physician and the estate, judgment being given for $10,000, at the same time giving the plaintiffs a judgment for the amount of their fees, $213.95.

From what I learned of this case, the patient did not die because of the x-ray injury, but in spite of it. However, some overdosage was given, which was the point in question. Because injury did occur, the doctors had to pay.

The case was appealed to the Court of Appeals of the State, and a rehearing was granted. At the second trial a judgment identical with the first was arrived at, and the case settled.

VERDICT AND A BIG JUDGMENT FOR A NORMAL SPINE

In a personal injury suit tried before the United States Circuit Court in Chicago, the defendant, a railway company, was sued by a "go-getter" female of uncertain age.

The plaintiff alleged that while riding in a passenger coach on defendant's train, the sudden stopping of the train caused her to be thrown so violently into the aisle and onto the floor of the coach, causing her to land on her back and thereby severely injure her spinal column, etc.

Roentgenograms showing an absolutely normal spinal column were examined by two physicians—professional witnesses—who have no standing as roentgenologists and who certainly know nothing regard-

ing the interpretation of roentgenograms. These two men testified that the films showed two fractured vertebral bodies.

A well-known Chicago roentgenologist of national reputation and the writer testified that the films showed a normal spine; but the jury brought in a verdict for the plaintiff and a judgment for $30,000.

Rather than continue the litigation the railway company settled the judgment for exactly one-fourth the amount the jury decided upon.

The foregoing recital is a far-too-often-seen example of how wealthy defendants have injustice done to them. This is one of the great wrongs of our present expert witness, jury, and court system, as discussed by the writer in RADIOLOGY, May, 1935.

DOCTOR THREATENED TO SUE LAWYER FOR MALPRACTICE

The saying that "if a man bites a dog, it is news," makes the following "near news," does it not?

An Illinois physician was sued by a former patient for malpractice. Defendant physician carried no malpractice insurance, but being a member of the State medical society, the suit was defended by the attorney for the State society.

At the trial, the jury brought in a verdict for the plaintiff, and judgment for a reasonable sum. Later the defeated defendant threatened the attorney with suit for *legal malpractice*, but evidently he reconsidered, as the suit has not been filed. Is not this a funny one?

HE WAS NOT GUILTY BUT HE PAID RATHER THAN STAND TRIAL

One of the oldest (in point of service) and most experienced roentgen therapists in this country, was sued for malpractice, mainly because of unkind and unfavorable comment of a famous dermatologist.

The plaintiff had a long-standing fissured eczema, with much scarring on both hands, and the defendant physician had applied only 7.5 ma.-min. of radiation, using a 2-inch spark gap, at 8 inches F.S.D. without filter to the eczematous areas, divided into five applications during forty days, in 1919.

The patient was an unusually thin skinned, pale blonde man, whose occupation necessitated his putting his hands into irritating solutions.

While the defendant knew and his insurance carrier was satisfied that he was absolutely and entirely innocent of having done any damage or produced any injury, and that no malpractice had occurred, it was deemed advisable and expedient to settle the case for $1500, which was one-tenth of the amount asked for in the suit. The chance that the jury of twelve, with an I.Q. of about ten years, egged on by a blatant shyster lawyer, might award almost any amount, was too great to risk. The case was one of the worst of the hold-up type, and was taken by the attorney purely for its nuisance value. This is the sort of thing that men who practise in the larger cities have to combat all the time. It is what makes our insurance rates so high.

It is very unfortunate for the defendants in medical malpractice or personal injury damage suits, that, in the trial of these cases, we are faced by a jury of twelve men or/and women, with various and varied human sympathies and (inhuman) prejudices, who are to be the umpires or referees as to whether we did or did not act correctly, and whether and how much we are to pay.

Often, aye, far too often, these jurors are horrified by the exhibition of scars, defects, and injuries, which are in themselves *due to the disease* which we have tried to relieve or cure, and which are played up to the greatest possible extent by the clever word-painting of the persuasive ability of the lawyers, for the purpose of creating bias, intolerance, and prejudice and eliciting sympathy and commiseration.

Another reason which seriously militates against us, in the trials of these suits, is the apparently increasing laxity of the courts, relative to the qualification and the qualifications of expert witnesses.

Many times in the writer's experience, witnesses have been put on the stand, have

been allowed to qualify as experts and have given expert testimony, when we knew that the so-called experts had absolutely no expert knowledge of the subject of their testimony. These men testify to what the attorneys want them to, if, as, and when they are employed and paid as such, and will do this day after day, without embarrassment or hindrance. This situation is one of the writer's pet peeves, and has been discussed by him, in season and out, perhaps with some beneficial effect; but probably not with any resultant good in the end.

COMPENSATION AND SETTLEMENT FOR INJURY BARS MALPRACTICE SUIT

Quite a number of State courts of last resort have decided that when an injured person has settled for his injuries, either in or out of court, he cannot maintain a damage suit against a physician for malpractice because of the same injury settled for by him with the *tort feasor* or party causing the original injury. Manifestly, this is as it should be, but every once in a while, some such instance as is described in the following incident comes up.

A medical confrère in a neighboring State wrote me that he was threatened with a malpractice suit by a man who had suffered a badly comminuted broken leg, in which the end-result was a considerable shortening and a slight irregularity of outline. Both bones of the leg were badly comminuted, and, in my friend's opinion, the result was good for the condition of the bones after the automobile had passed over them. Quoting from the letter:

"The patient was a truck driver, who had just alighted from his driving seat, more or less under the influence of liquor, reeled against an electric car that was just starting and was thereby pushed toward a moving automobile, the front wheel of which passed completely over his leg.

"Even before the patient was walking upon the injured leg, he started suit against the owner of the automobile and against the electric railway company. Because he had a big lawyer, who nearly always won his cases, these two defendants joined in a settlement, paying the truck driver $9,000 and, of course, securing a release.

"Now, after nearly two years, this patient is threatening to sue me for malpractice, because his leg is shorter than it originally was and not absolutely straight, and probably mostly because I am trying to collect my fee.

"I have no insurance against malpractice suits and do not want to lose my fee, as I feel that having rendered him good service and saved a useful leg for the skalliwag, I should be paid.

"Thanking you for such advice and information as I know you are able to give, I am, Fraternally, . . . "

I replied as follows:

"My Dear Hal:—Your interesting letter of the 7th received yesterday. I cannot help smile and say, 'I told you so.' You may remember that only a few months ago you said that you did not need malpractice insurance, and now you are almost ready to weep, just because a rascally dead beat is threatening you. May I now say that if you had insurance against that sort of thing, you would have no need for worry, and that, not having any, you may have to pay a lawyer more than insurance would cost you for from two to four years.

"However, I feel pretty sure that you have nothing much to worry about. If that scamp has a good lawyer—and you say he had in the damage suits—you may not even need to employ a lawyer of your own.

"This is what you are to do. Go to that patient's lawyer and tell him that you know that neither he nor anyone else has one chance in a hundred of securing a verdict against you, because of the fact that his client *has already been paid for the damages done by the automobile* that ran over his leg. That any shortening or slight deformity which might be present at this time is the direct result of the original injury, that the defendants in the first suit acknowledged and paid for the injury, and that you are amply ready and able to prove that the foregoing is true. Tell him that you can produce plenty of expert testimony that the end-result of your treatment is as good

as can be expected, and furthermore, that you want to be paid for your services.

"If, after that, the lawyer still insists upon suing you—and I very much doubt that he will—go to I . . . C. . . . and see Mr. W. B. D. . . . . ., tell him your story and show him this letter. He knows more about defending medical malpractice suits than any man in that section, or anywhere for that matter, and I am sure will clear you.

"Another way to handle this affair is to see D. . . . . first and have him communicate with your patient's lawyer. It may cost you fifty or a hundred dollars for D. . . . .'s services, but it will be money well spent.

"If you have Mr. D. . . . . in on the case, do not sue for your fee unless he (D.) says you should. In the meantime, find out where your patient does his banking, so that if it is necessary you can attach his bank account if you want to.

"In a recent case in Oklahoma [Alexander *vs.* Wedel, 37 Pac. R. (2), 252] this same sort of proposition was under consideration. A man sustained an injury consisting of fractures of bones in the arm and forearm, necessitating amputation of the arm above the elbow. The Industrial Commission very properly awarded him compensation for full loss of his arm. Alleging that the loss of his arm was due to negligence and unskillfulness of the attending physician and the hospital, the injured man brought suit (after the Industrial Commission settlement) for malpractice. The trial court found for the defendants and on appeal the Supreme Court of Oklahoma said that it had definitely decided that question before. It further said that 'a worker is entitled to receive compensation for an injury sustained in the course of his employment, and that such award included compensation for the original injury, the ultimate results of such injury, including the carelessness or negligence of the physician or hospital selected by the employer, as well as the suffering and mental anguish sustained.'

"The Oklahoma Supreme Court therefore sustained and affirmed the judgment of the lower court."

My friend went to see the patient's lawyer, told him what I had advised him to say, and wrote me shortly afterward, that he "had the pleasure of learning that the lawyer had told his client identically the same thing." The fee was paid at a slight reduction and everyone was happy. *That physician immediately bought malpractice insurance.*

NO! THANK YOU!

A few years ago I received a letter from a firm of "counsellors-at-law" in a large city within a hundred miles of Chicago, in an adjoining State, which read as follows:

"As attorneys for S. . . . N. . . . , we started a malpractice suit against Dr. D. K. Q. . . . because of an x-ray burn sustained by Mr. N., through which he lost the use of his left hand. Dr. Q., against whom this suit is brought, lost the use of three of his fingers through burns sustained by him at the same time.

"Drs. I. G. and I. C. Q. of this city recommended that we communicate with you for the purpose, if possible, of retaining you as an expert witness rather than use the local doctors. Will you therefore please advise us whether or not you are licensed to practice in the State of . . . . ., because under our Statutes, to qualify as an expert witness, it is necessary that a doctor from without the State be licensed to practise in this State. Also please give the conditions under which you will come."

Having heard about this suit from another source and not caring to get mixed up in it, under any circumstances, I promptly answered: "Replying to your letter of yesterday, will say that I am not at this time licensed to practise in your State."

From previous knowledge I knew that the two physicians (in the same city) named by the attorneys had declined to appear against the defendant in the suit, and gave my name, knowing that I would be able to wiggle out of testifying.

I had previously conferred with two attorneys relative to aiding in the defense of these suits (there were two suits, one of

them being the one mentioned above, and the other brought by the employer of the plaintiff, to recover the amount of their liability to him).

The defendant physician had used a hand fluoroscope in trying to locate a piece of steel in the plaintiff's hand, and had kept the energized tube active so long that he had injured his own hands so as to necessitate the amputation of three fingers.

These two cases were settled for $4,300.

EXPERT TESTIMONY RELATIVE TO THE KNEE JOINT, ETC.

A colleague in a neighboring State wrote me: " . . . I have on several occasions been called to testify in compensation cases, and the question arose as to whether or not one can testify truthfully and honestly with regard to an injury of the knee joint involving rupture of the semilunar or cruciate ligaments, that one can tell definitely by means of an x-ray picture if such injuries to said ligaments are present. Authorities on this subject differ and certainly it leaves one in an embarrassing situation to see another M.D. under oath, sit before a judge or arbitrator and testify that in his opinion the x-ray that he took himself plainly shows an injury to either cruciate or semilunar cartilage. All this, when at the same time the nature of the injury, together with the clinical findings and follow-up of the case, are all against such diagnosis. Yet during these trials, one has to sit and listen to an unethical M.D. testify that certain injuries exist *because the x-ray pictures shows that injury* and at the same time cites—at random—authorities claiming that the x-ray will show and does show such injuries.

"I have a high regard for your opinion and wish you would set me clear on this point, so that we may definitely refute and deny this man's appearance of superior knowledge, when we absolutely know that he is a blatant four-flusher."

The writer of the above is a ruggedly honest, sincere surgeon, with a high sense of honor, good reputation, and more than usual ability. My reply was in part as follows: "Your query as to 'whether or not one can testify truthfully and honestly with regard to an injury to the knee joint, involving rupture of the semilunar or *cruciate* ligaments, that one can tell definitely by means of an x-ray such injuries to said ligaments are present,' seems to me to need some clarification before answering. First the semilunar structure, which you undoubtedly mean, is the semilunar cartilage, and not in any sense a ligament, as I understand ligaments. And second, you must mean the *crucial* ligament instead of the *cruciate*, as the crucial is in the knee, while the cruciate is in the neck.

"Now then, with these two items straightened out, the answer is that, under absolutely ideal conditions, with a thin, bony (neither fat nor muscular) knee, exactly the correct technic in making the exposure, perfect dark room technic, and the consequent production of a 100 per cent perfect roentgenogram, a semilunar cartilage *may* be shown, seen, and diagnosed. Understand me, Doctor, *theoretically this is possible but it is far from probable.* I have seen and examined many hundreds of injured knees and have studied more than that number of roentgenograms of knees, and as far as I can remember, I have seen, shown, and correctly diagnosed two dislocated semilunar cartilages, which were of recent occurrence.

"Dr. George W. Holmes, roentgenologist at the Massachusetts General Hospital and an international authority on roentgen diagnosis, says: 'With good films taken in the anteroposterior and lateral positions, it is usually possible to visualize the line of fracture, and to determine whether or not it involves the joint. *Cartilage is not of sufficient density to be distinguishable from soft tissue, and injuries involving such, as displaced semilunar cartilage, or separation of the articular cartilage from the condyles, are not demonstrable, except when a small fragment of bone has accompanied the displaced cartilage, or cases of long duration where calcification (of the cartilage) has occurred.*'

"Regarding the crucial ligaments, a rupture of either (or both in the same knee), *cannot be diagnosed by roentgenograms* and I defy anyone to even present an argument why these should show or be shown or be possible of diagnosis *per se*. Of course if the tibial spine is fractured, one may take for granted that one or both of the crucial ligaments may either be torn or displaced, because these are attached to the anterior and posterior aspects of the anterior and posterior tibial spines, respectively, and when torn loose, may have fragments of the bone attached, and *these bony fragments may be seen*. Now, if, in such a case, the tibial spines are seen to be missing or deformed as though from fracture, it requires for the observer only to be able to add two and two together to make the classical four, and in that way make a diagnosis—yes, even a correct diagnosis, under the conditions just stated.

"But, as I said, *laceration, rupture, or tearing of the crucial ligaments* per se, *without accompanying fracture of the bony attachments, cannot be diagnosed by means of the roentgen rays or any other means except open inspection of the parts.*

"When such a man as you have there, testifies as you say, why do not you, or someone who is interested, try to secure the opinion of someone in whom you have confidence, in that particular instance. This can be done, either before the trial or hearing, or if after the trial, apply for a new trial or appeal the case if new trial is refused, and then secure the expert's opinion on the film or films. Then if the expert agrees with you or whoever is interested, have him appear at the next trial of the case and interpret the films correctly. That is the way it is done here, and pretty often those 'wise guys' who can see almost anything, and much more than anyone else, in their own roentgenograms, draw in their horns. That is the only way I know of to correct this growing evil. We have a lot of that same sort of thing, in instances in which the same man qualifies as an x-ray expert in one case, a neurologist in another, a pediatrician in another, and as a pathologist or some other specialty the next day. I have actually seen one man qualify as an expert in three widely separated medical specialties on the same floor of our Cook County Court House, in the same morning. Incidentally, that same doctor has been barred from giving testimony as an expert witness, in some of our municipal and superior courts. The crooked rascal should have been debarred years ago. He would testify any way he could get paid, would swear that black was white for ten dollars, and could see more fractures than any other man in the county.

"Well, have I answered your query? If not, let me know, and I will try again. I get wound up on such things as these, and feel as though I should fight all the battles over again, so excuse my verbosity and accept it as it is meant, to be helpful."

DULCE EST DESIPERE IN LOCO

Being considered somewhat a practical joker and having a fairly well-developed sense of humor, it has pleased me at times to appear to be stupid and slow thinking.

Several times, that line of conduct has served to make it easy for me to avoid apparent embarrassment and aggravation, and at the same time perpetrate a joke or turn a laugh upon some one who is trying to appear smart or witty.

While on the witness stand, in a personal injury suit trial, wherein my direct testimony had been particularly damaging to the defendant in the case, I was asked, during cross-examination, "Doctor, have you ever appeared as an expert witness against the . . . Company in a suit before?" After I had replied in the affirmative, the lawyer, pugnaciously and combatively barked out, "What suit?" To his surprise and apparent disgust, I meekly replied, "I am not sure, but I think it was my blue serge suit."

That lawyer handled me very carefully after that, and did not make any further attempts to press the point about what suits I had appeared in.

(*To be continued*)

STUDY OF BACK-SCATTERED RADIATION AND DEPTH DOSAGE USING A TRANSPLANTABLE ANIMAL TUMOR AS AN INDICATOR

By KANEMATSU SUGIURA, D.M.Sc., *New York City*

From the Huntington Fund for Cancer Research, Memorial Hospital, New York

WHEN a beam of radiation strikes matter, some of the rays are scattered in all directions. Among other factors, the amount of scatter depends upon the quality of the radiation, the nature of the material upon which it impinges, the volume of the material, and the area of the beam. The relative amount of scattered radiation is different in different directions, being greatest in the forward and least in the backward direction (1).

Many investigators have measured the scattered radiation by means of ionization chambers, photographic films, and various chemical and biologic media, placed upon the surface of or in water, paraffin, or the human body. The use of different materials combined with variations in experimental conditions has led to varying results and, therefore, much controversy.

The work here reported was planned to determine the back-scattered radiation and depth dosage by means of tumor tissue at different depths in a block of paraffin. It is possible that the results of this study may throw some light upon the existing differences among physical, chemical, and biologic measurements of radiation.

STUDY OF BACK-SCATTERED RADIATION

In a previous study (2) on the reaction of transplantable Mouse Sarcoma 180 to radiation of different wave lengths, we found that the radiosensitivity of this tumor to filtered roentgen rays was constant from time to time and that the change in the viability of the transplants was definite and easily measurable. Furthermore, the reaction was not influenced by slight changes in laboratory conditions, or other extraneous causes. In addition, the quantity of radiation required to produce the measurable changes was in the neighborhood of the amounts ordinarily used in therapy.

Accordingly, in the present experiments a number of observations were made with Mouse Sarcoma 180 irradiated in air to determine whether or not the radiosensitivity of the tumor had remained the same as had previously been determined.

In order to avoid uncertainties due to secondary radiations and the loss of tissue water, a special cellophane chamber was constructed. One layer of very thin cellophane (0.025 mm.) was stretched over an ordinary wooden embroidery hoop, with a diameter of 9.5 centimeters. On this was placed a piece of filter paper (0.13 mm.), slightly moistened with a Locke-Ringer solution at PH 7.0. Fragments of fresh tumor tissue, each weighing about 6 mg., and measuring 1.5–2.0 mm. in thickness, were then arranged in rows on the paper. A second embroidery hoop with a diameter of 12 cm., with one sheet of cellophane stretched across, was then placed over the first, to reduce evaporation and prevent bacterial contamination. Aseptic precautions were taken throughout.

In the present experiments, the tumor fragments were subjected to radiation from a standard water-cooled Coolidge roentgen tube under the following conditions: 200 kv., 32 ma., 0.5 mm. copper and 1.6 mm. aluminum filter, 50 cm. distance and 400 sq. cm. field. Under these conditions the tumor tissues received 51.5 r per minute.

At the end of definite intervals of time, the irradiated tumor fragments were removed from the cellophane moist chamber and immediately implanted into healthy white mice in the usual way, or they were transferred temporarily to a small Petri dish containing a sheet of moist filter paper and allowed to remain in the re-

frigerator until ready for implantation (always within less than two hours). The behavior of the transplantable tumor and method of recording the development of the irradiated and non-irradiated tumor tissues in the host have been reported elsewhere.

The results obtained from this procedure showed that 25 minutes' exposure (about 1,290 r) of the tumor fragments to radiation prevented 10 per cent of them from growth. As irradiation continues the tumor cells die quickly. At 35 minutes' exposure (about 1,800 r), and 45 minutes' exposure (about 2,300 r), marked inhibition and delayed growth were observed, the percentage of inhibition being 40 and 70, respectively. However, the viability of the transplants was completely destroyed by 55 minutes' exposure (about 2,800 r). These figures are essentially the same as those of the earlier experiments (1933), indicating that results are reproducible when approximately the same experimental conditions are used. It may be stated that during the period of 20 months the radiosensitivity of Mouse Sarcoma 180 has not altered. These findings indicate that this is a good material for the investigation of radiologic problems.

For the study of surface and depth doses in radiation therapy, various scattering materials, such as the cadaver, animal tissues, and phantoms constructed of water, paraffin, beeswax, wood, and grain, have been used. Since in doing this work the object in view was to obtain data which could be used for dosage purposes in roentgen therapy, it would be desirable to use the tissues of the body. Obviously, however, living human tissue could not be used to experiment upon. Water, although it has a density fairly close to the tissues of the human body, is not a convenient material. It was decided, therefore, to use a paraffin phantom, which while not representing closely the human body, is satisfactory for the comparison of results with other methods. This is particularly true in our case since members of the

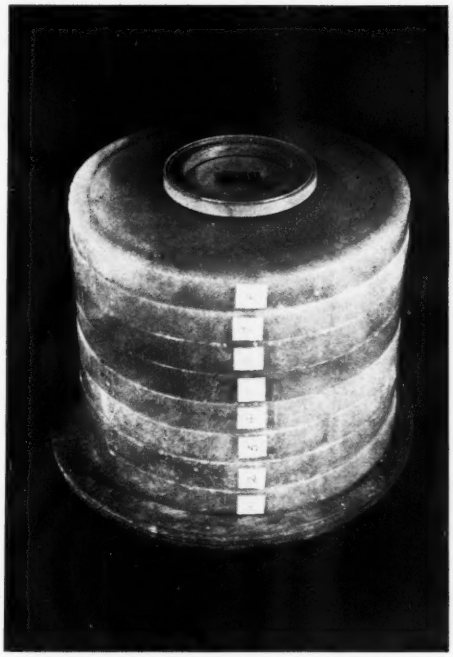

Fig. 1. Showing the position of a cellophane moist chamber at the surface of a paraffin phantom for exposing tumor tissues to 200 kv. roentgen rays. The fragments in this photograph are those of the Passey mouse melanoma.

Memorial Hospital Staff had employed a similar paraffin phantom in their physical and biologic measurements of back-scattered radiation and of depth dosage, using various ionization chambers, *Drosophila* eggs, and wheat seedlings.

The arrangement of the test material for exposure to 200 kv. roentgen rays is shown in Figure 1, and diagrammatically in Figure 2. The phantom consists of eight slabs of paraffin, having a diameter of 30 cm. and a thickness of about 3 centimeters. Each of the top four slabs had a shallow cavity in the center, 10 cm. in diameter and about 5 mm. in depth. In the topmost one, as shown in Figure 2, the floor of the same cavity was 2 cm. below the surface. The distance between the target and the surface of the paraffin phantom was 50 centimeters. The distances between the target and the floors of the cavities of the paraffin slabs were 52, 53.6, 56.7, 59.8, and 62.9 cm., respectively.

These cavities were plugged temporarily with paraffin discs according to the requirements of the experiment.

proliferating capacity of sarcoma cells measured in air.

In order to determine the effect produced

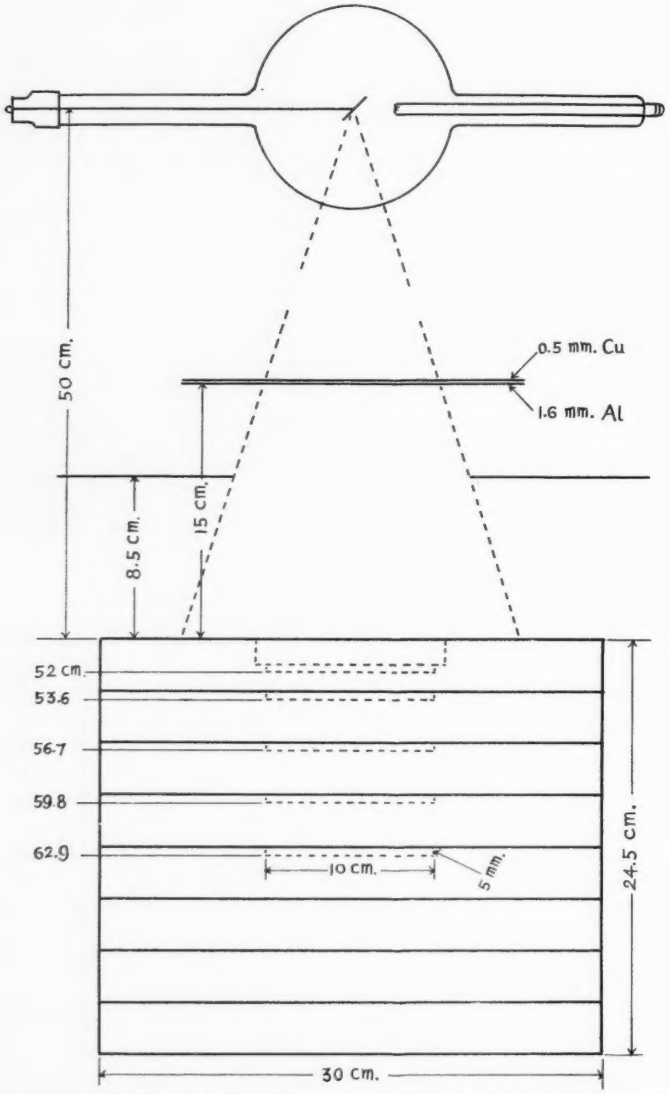

Fig. 2. Diagram of paraffin phantom, showing the position of the shallow cavities with respect to the diaphragm, filters, and source of radiation.

The size of the beam reaching the paraffin phantom was regulated by a lead diaphragm. It was the same as that used in the previous study in determining the destructive action of roentgen rays on the

by the back-scattered radiation, the cellophane moist chamber containing the fragments of fresh Mouse Sarcoma 180 was placed in the surface of the paraffin phantom, the cavities in which had been filled

with their respective discs, as shown in Figure 1, and subjected to radiation under the conditions previously described. The scatter from the paper and cellophane is negligible.

At the end of definite intervals of time, the irradiated tumor fragments were removed from the cellophane moist chamber and implanted into animals in the usual way. The results of this series of experiments are presented in Table I. In the last column we have given a brief account of the behavior of the irradiated and non-irradiated tumors. In Figure 3 the extent of tumor takes in percentage is plotted against the time of treatment in minutes. In this figure a graphic comparison is given between the values obtained from irradiating tumor fragments in air and on the surface of paraffin, other conditions being identical. The curve *A* was plotted from data obtained in previous experiments (2), and verified by four observations (indicated by *X*) made at the same time at which the observations recorded on the curve *B* were obtained.

From these curves it will be seen at once that the injurious action of filtered roentgen rays on the proliferating capacity of sarcoma cells was decidedly increased when the tumor tissues were irradiated

TABLE II.—RATIO OF INHIBITORY EFFECT IN AIR TO INHIBITORY EFFECT ON SURFACE OF PARAFFIN PHANTOM FOR SAME FIELD (BACK-SCATTERING)

Per cent of Tumor Takes	Air (Min.)	Paraffin Phantom (Min.)	Percentage of Radiation Effective on Surface
80	31	24	129
70	34	26	131
60	37	28	132
50	39	30	130
40	42	32	131
30	44	34	129
20	47	37	127
10	51	40	128
Average (percentage of radiation effective on surface)			130

TABLE I.—RESULTS OF TRANSPLANTING MOUSE SARCOMA 180, AFTER EXPOSURE TO FILTERED ROENTGEN RAYS (200 KV., 32 MA., 0.5 MM. CU + 1.6 MM. AL FILTER, 50 CM. DISTANCE, 400 SQ. CM. FIELD) AT THE SURFACE OF PARAFFIN PHANTOM

Exp. No.	No. Tumor Transplants Used	Duration of Exposure (Min.)	Growth of Transplants (Per Cent)	Remarks
1	10	Controls	100	Rapid growth
	10	10	100	Rapid growth
	10	20	80	Two tumors did not grow; 2 tumors grew slowly; others grew rapidly
	10	30	50	Five tumors did not grow; 3 tumors grew slowly; others grew rapidly
	10	40	10	Nine tumors did not grow; 1 grew slowly
	10	50	0	No growth
2	10	Controls	100	Rapid growth
	10	5	100	One tumor grew slowly; others grew rapidly
	10	25	60	Four tumors did not grow; 2 tumors grew slowly; others grew rapidly
	10	45	0	No growth
	10	55	0	No growth
3	10	Controls	100	Rapid growth
	10	15	100	Rapid growth
	10	20	90	One tumor did not grow; 2 tumors grew slowly; others grew rapidly
	10	30	60	Four tumors did not grow; 2 tumors grew slowly; others grew rapidly
	10	35	30	Seven tumors did not grow; others grew slowly
4	20	Controls	100	Six tumors grew slowly; others grew rapidly
	20	35	30	Fourteen tumors did not grow; others grew very slowly
	20	40	20	Sixteen tumors did not grow; others grew very slowly
	20	45	0	No growth

TABLE III.—RESULTS OF TRANSPLANTING MOUSE SARCOMA 180, AFTER EXPOSURE TO FILTERED ROENTGEN RAYS (200 KV., 32 MA., 0.5 MM. CU + 1.6 MM. AL FILTER, 400 SQ. CM. FIELD) IN PARAFFIN PHANTOM AT DIFFERENT DEPTHS

Exp. No.	No. Tumor Transplants Used	Duration of Exposure (Min.)	Depth (Cm.)	Growth of Transplants (Per Cent)	Remarks
1	10	Controls	..	100	Rapid growth
	10	45	0	0	No growth
	10	45	2	0	No growth
	10	45	3.6	20	Eight tumors did not grow; others grew very slowly for 3 weeks, normal thereafter
	10	45	6.7	20	Eight tumors did not grow; others grew very slowly for 3 weeks, normal thereafter
	10	45	9.8	80	Two tumors did not grow; others grew slowly for 2 weeks, normal thereafter
	10	45	12.9	90	One tumor did not grow; 6 tumors grew slowly for 2 weeks, normal thereafter; others grew rapidly
2	10	Controls	..	90	One tumor regressed after growing for 2 weeks; 7 tumors grew slowly; others grew rapidly
	10	45	0	0	No growth
	10	45	3.6	10	Three tumors did not grow; 6 tumors regressed after growing for 2 weeks; 1 grew very slowly
	10	45	6.7	40	Four tumors did not grow; 2 tumors regressed after growing for 2 weeks; others grew very slowly
	10	60	6.7	10	Six tumors did not grow; 3 tumors regressed after growing for 2 weeks; 1 grew very slowly
	10	90	9.8	0	Six tumors did not grow; 4 tumors regressed after growing for 2 weeks
	10	90	12.9	0	Eight tumors did not grow; 2 tumors regressed after growing for 2 weeks
	10	100	12.9	0	Eight tumors did not grow; 2 tumors regressed after growing for 2 weeks
3	10	Controls	..	100	One tumor regressed after growing for 3 weeks; 2 tumors grew slowly; others grew rapidly
	10	45	3.6	0	No growth
	10	45	6.7	10	Nine tumors did not grow; 1 tumor grew very slowly
	10	45	12.9	100	Five tumors grew slowly for 2 weeks, normal thereafter; others grew rapidly
	10	90	9.8	0	Eight tumors did not grow; 2 tumors regressed after growing for 2 weeks
	10	90	12.9	10	Eight tumors did not grow; 1 tumor regressed after growing for 2 weeks; 1 tumor regressed after growing for 4 weeks
4	10	Controls	..	100	Rapid growth
	10	5	6.7	100	Rapid growth
	10	10	6.7	100	Rapid growth
	10	20	6.7	100	Four tumors grew slowly; others grew rapidly
	10	30	6.7	70	Three tumors did not grow; 6 tumors grew slowly; 1 tumor grew rapidly
	10	40	6.7	30	Seven tumors did not grow; others grew slowly
5	10	Controls	..	100	Two tumors grew slowly; others grew rapidly
	10	40	6.7	10	Nine tumors did not grow; 1 tumor grew very slowly for 3 weeks, normal thereafter
	10	50	6.7	10	Nine tumors did not grow; 1 tumor grew very slowly for 3 weeks, normal thereafter
	10	60	6.7	0	No growth
	10	70	6.7	0	No growth
6	20	Controls	..	100	Rapid growth
	20	80	6.7	0	No growth
	20	100	9.8	0	No growth
	20	120	12.9	0	No growth
7	10	Controls	..	100	Rapid growth
	10	25	9.8	100	Two tumors grew slowly for 2 weeks, normal thereafter; others grew rapidly
	10	35	9.8	100	Four tumors grew slowly for 2 weeks, normal thereafter; others grew rapidly

TABLE III.—*Continued*

Exp. No.	No. Tumor Transplants Used	Duration of Exposure (Min.)	Depth (Cm.)	Growth of Transplant (Per Cent)	Remarks
	10	40	9.8	90	One tumor did not grow; 2 tumors grew slowly for 2 weeks, normal thereafter; others grew rapidly
	10	50	9.8	50	Five tumors did not grow; 2 tumors grew slowly; 3 tumors grew slowly for 2 weeks, normal thereafter
7	10	80	9.8	10	Nine tumors did not grow; 1 tumor grew very slowly
8	10	Controls	..	100	Rapid growth
	10	45	9.8	90	One tumor did not grow; 5 tumors grew slowly; others grew rapidly
	10	60	9.8	40	Six tumors did not grow; 3 tumors grew slowly; 1 tumor grew rapidly
	10	75	9.8	30	Seven tumors did not grow; others grew very slowly
	10	90	9.8	0	No growth

upon the surface of the paraffin. Thus the viability of the transplants was completely destroyed by 45 minutes' exposure, whereas when tumor tissues were exposed to radiation in the air, it required about 57 minutes' exposure. To obtain 50 per cent inhibition, the former required 30 minutes' exposure and the latter 39 minutes' exposure.

To obtain the percentage of back-scatter, Table II was prepared from Figure 3. In Table II, Column 2, are given the dosages in length of exposure expressed in minutes, as obtained in the air for different percentages of tumor takes. Similar values obtained at the surface of a paraffin phantom are given in Column 3. Dividing the values in Column 2 by those in Column 3, respectively, we obtain the figures in Column 4, which represent the relative amounts of radiation affecting the tumor fragments on the paraffin as compared to 100 for the tumors in free air. It will be noted that these ratios differ very little throughout the range, so the average value of 130 is quite accurate for this biologic material. The percentage back-scatter as usually expressed is the difference between the figures of Column 4 and 100. Or, taking the average value of 130, the back-scatter as determined in these experiments is 30 per cent.

It is interesting to compare our results with those obtained by Mrs. Quimby at the Memorial Hospital. She measured the back-scattered radiation with a mesh ionization chamber and a small spherical celluloid ionization chamber, using a similar paraffin phantom. The percentage of back-scatter obtained with a 400 sq. cm. field and the same quality of radiations using the "mesh" chamber is 78, and, for a small spherical celluloid ionization chamber under the same conditions, it is 55 as compared to 30 for our tumor fragments. It will be seen, therefore, that there is no very close agreement between the results of these physical and biologic determinations. The difference in these results is difficult to account for. On the other hand, the present results are in a close agreement with the findings of Packard (3) who, working with *Drosophila* eggs, found a percentage of back-scatter of 37. In his experiments, the *Drosophila* eggs were placed on the surface of a solid mass of paraffin and were irradiated under the following conditions: 190 kv., 0.5 mm. copper and 1 mm. aluminum filter, and 300–400 sq. cm. field.

STUDY OF DEPTH DOSAGE

In the radiation treatment of malignant neoplasms, it is essential to know not only the quantity of radiation delivered to the tissue at the surface of the body, but also

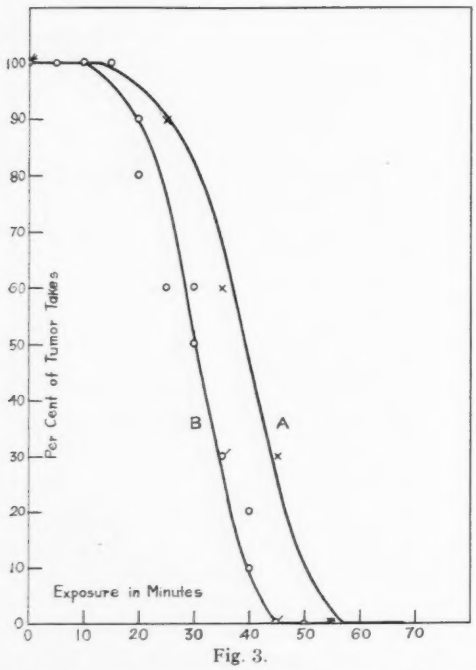

Fig. 3.

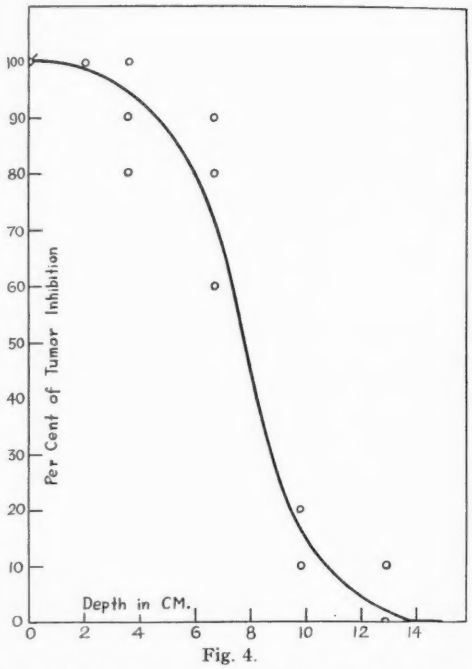

Fig. 4.

Fig. 3. Survival curves of Mouse Sarcoma 180, exposed to 200 kv. roentgen rays, filter 0.5 mm. Cu + 1.6 mm. Al, 400 sq. cm. field, intensity = 51.5 γ/min. at 50 cm. in air. Each point represents ten or more tumor fragments. Each line radiating from a point indicates an additional identical point.

A = in the air.

B = on the surface of paraffin phantom.

Fig. 4. Curve showing variation in tumor inhibiton at different depths in paraffin phantom for 200 kv. roentgen rays, 0.5 mm. Cu and 1.6 mm. Al filter, 50 cm. target-surface distance, 400 sq. cm. field, and 45 minutes' exposure.

the depth dose within the body. Transplantable animal tumors afford good material for such a study.

As in the case of the preceding experiments, the fragments of fresh Mouse Sarcoma 180 were arranged in rows on a piece of moistened filter paper placed in the shallow cavity at various depths in the paraffin phantom (Fig. 2). To avoid the loss of moisture, one sheet of very thin cellophane was stretched across the top of the shallow cavity of each paraffin slab. Tumor fragments were placed at the various depths in the phantom and the latter was placed directly under the target with the surface at 50 cm. therefrom. The conditions of irradiation were the same as in the previous experiments. Observations were made at depths of 0 (surface), 2, 3.6, 6.7, 9.8, and 12.9 centimeters. The temperature of the roentgen-ray chamber during the irradiation was 22° to 26° Centigrade. At the end of definite intervals of time, some of the irradiated tumor fragments were removed from the paraffin slabs and inoculated into mice. The results of this series of experiments are presented in Table III. The data are arranged according to the increasing depth at which tumor fragments were irradiated.

From the data in Table III, it will be seen that tumor fragments exposed to radiation for from 5 to 20 minutes at the 6.7 cm. depth, 25 to 35 minutes at 9.8 cm. depth, or 45 minutes at 12.9 cm. depth remained viable, the growths being practically the same as in the non-irradiated controls. As irradiation continues, the lethal effect on the tumor cells increases. The viability of the transplants was completely destroyed by the radiation at the

depths of 6.7, 9.8, and 12.9 cm., in 65 minutes, 90 minutes, and 100 minutes of exposure, respectively. The graphic representation of Figure 4 shows the extent of tumor inhibition in percentage at various depths, the time of exposure being the same in all cases, *i.e.*, 45 minutes.

In the determination of tissue doses, it is necessary to know (1) the amount of radiation falling on the skin, and (2) the relative amounts reaching various depths in the tissues. Figure 5 shows the survival curves of Mouse Sarcoma 180 exposed to 200 kv. roentgen rays at the surface of the paraffin phantom, and within the paraffin phantom at the depths of 6.7 and 9.8 centimeters. The extent of tumor takes in percentage is plotted against time of treatment in minutes. To obtain the percentage depth doses, Table IV was prepared from these curves. In Table IV, Column 2, are given the dosages in length of exposure to the filtered roentgen rays, expressed in minutes obtained at the surface of the paraffin phantom for different percentages of tumor takes. Likewise the values obtained at depths of 6.7 and 9.8 cm. in the paraffin phantom are given in Columns 3 and 4. Dividing the values in Column 2 by those in Column 3 or 4, respectively, we obtain the figures in Column 5 or 6, which represent the percentage depth dose for the depths indicated.

It is interesting to compare our results with those obtained by other experimenters at the Memorial Hospital, who have measured the depth dose by means of ionization chambers and various biologic media, using a similar paraffin phantom under the same conditions of irradiation. The percentage depth doses at 6.7 and 9.8 cm., determined with a small spherical celluloid ionization chamber under comparable conditions, are 72 and 54, respectively, as compared to 86 and 54, respectively, for our experiments. It will be seen, therefore, that there is a very close agreement in the results of these physical and biologic determinations at the depth of 9.8 centimeters; but the value obtained at the depth of 6.7 cm. by the biologic method is somewhat higher than that by the physical method. The difference in the results may be due to experimental error.

TABLE IV.—RATIO OF INHIBITORY EFFECT ON SURFACE OF PARAFFIN TO INHIBITORY EFFECT IN PARAFFIN PHANTOM AT VARIOUS DEPTHS (DEPTH DOSES)

Per Cent of Tumor Takes	At Surface (Min.)	At 6.7 Cm. Depth (Min.)	At 9.8 Cm. Depth (Min.)	Percentage of Depth Dose	
				6.7 Cm.	9.8 Cm.
80	24	28	47	86	51
70	26	30	49	87	53
60	28	32	52	88	54
50	30	34	55	88	55
40	32	37	59	86	54
30	35	40	64	88	55
20	38	45	70	84	54
10	42	50	78	84	54
Average (percentage of depth dose)				86	54

Figure 6 shows the percentage depth dose curve determined with a small spherical celluloid ionization chamber for 200 kv. roentgen rays in the paraffin phantom at various depths. The percentage depth doses determined with a transplantable tumor under comparable conditions at various depths are indicated by circles. The points at 2, 3.6, and 12.9 cm. depths are obtained from the data in Table III, using similar means of obtaining the depth dose values as those used for depths of 6.7 and 9.8 centimeters. Since the number of experiments performed at the depths of 2, 3.6, and 12.9 cm. is small, these points on the curve are not known with the same degree of precision. At any rate, within reasonable limits it will be seen that there is a close agreement between the results of these physical and biologic determinations, the latter giving somewhat higher values.

It may be of interest to mention that the 112 tumors which survived the irradiation at various depths in the paraffin phantom, the time of exposure being from 20 to 90 minutes, 71 tumors, or 63 per cent, grew at much below the normal rate, and only 41 tumors, or 37 per cent, showed rapid growth. In some cases the retarded

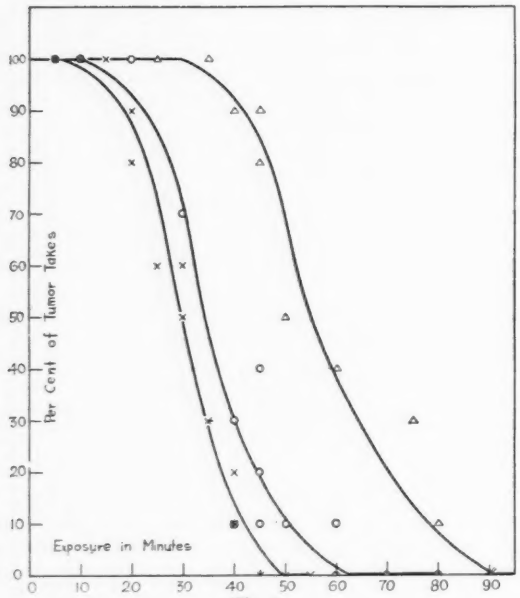

Fig. 5.

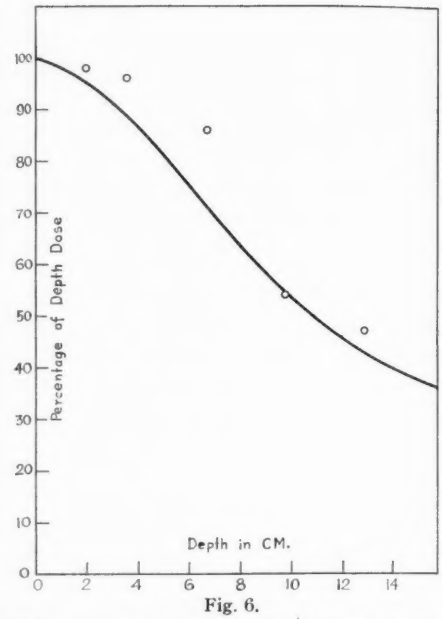

Fig. 6.

Fig. 5. Survival curves of Mouse Sarcoma 180, exposed to 200 kv. roentgen rays, filter 0.5 mm. Cu + 1.6 mm. Al, 400 sq. cm. field, 50 cm. target-surface distance, at the surface of and in a paraffin phantom at the depths of 6.7 and 9.8 centimeters. Each point represents ten or more tumor fragments. Each line radiating from a point indicates an additional identical point.

X = on the surface of paraffin phantom.

O = at 6.7 cm. depth.

Δ = at 9.8 cm. depth.

Fig. 6. Curve showing depth doses determined with a small spherical celluloid ionization chamber for 200 kv. roentgen rays, 0.5 mm. Cu + 1.6 mm. Al filter, 50 cm. target-surface distance and 400 sq. cm. field, in paraffin phantom at various depths.

O = depth doses determined with a transplantable tumor under comparable conditions at various depths.

tumor growth continued for several weeks, and the tumor subsequently underwent complete regression, while in the others retardation lasted only from two to three weeks and growth was normal thereafter. Furthermore, under the stated conditions of irradiation, no stimulating influence upon Mouse Sarcoma 180 has been noted.

SUMMARY

(1) The amount of back-scattered radiation from paraffin and the depth doses delivered in a block of paraffin at different depths for 200 kv. roentgen rays, 0.5 mm. Cu + 1.6 mm. Al filter, 50 cm. target-surface distance and 400 sq. cm. field, have been measured by a biologic method employing transplantable tumors as an indicator.

(2) The back-scatter obtained by this method is 30 per cent.

(3) The corresponding depth doses for the paraffin phantom are 86 per cent and 54 per cent for depths of 6.7 and 9.8 cm., respectively. These values agree reasonably well with the depth doses determined by ionization measurements made under similar conditions.

(4) About three-fourths of the tumors which survived the roentgen radiation at the surface of or in a block of paraffin grew at much below the normal rate when the tumor fragments received a dose greater than about 60 per cent of the full lethal dose. Under the stated conditions of irradiation, no stimulating influence upon Mouse Sarcoma 180 has been noted.

I wish to express my thanks to Dr. G.

Failla for his co-operation and helpful suggestions during the course of the present study.

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RADIATION THERAPY

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RADIATION therapy is the therapeutic application to the human body of all wave lengths of the electromagnetic spectrum, from the Hertzian waves on one end of the spectrum to the short gamma rays of radium on the other end. For practical purposes, the most important of these waves are infra-red, ultra-violet, Grenz, roentgen, and radium rays; our chief interests for purposes of this discussion are roentgen and radium rays.

The electromagnetic spectrum may be described by expressing the wave length of the various rays in Ångstrom units or in millimicrons, the latter being the method used by the United States Bureau of Standards. One Ångstrom unit, Å.U. or Å., equals one-tenth millicron, or one ten-millionth millimeter. One millimicron, Mμ or $\mu\mu$, equals ten Ångstrom units, or one-millionth millimeter. The wave lengths of the various rays are as follows: Hertzian waves, from 15,000 millimicrons to several kilometers; infra-red rays, from 770 to 15,000 millimicrons; visible rays, violet, indigo, blue, green, yellow, orange, and red, from 390 to 770 millimicrons; ultra-violet, from 13.6 to 390 millimicrons; Grenz or borderline rays are situated between ultra-violet and roentgen rays. The zone cannot be sharply bounded, but, as closely as can be determined, electromagnetic oscillations from about two Ångstrom units produce unique clinical and biological manifestations. Wave lengths of roentgen rays range from 0.14 to 13.6 millimicrons, and of gamma rays, from 0.001 to 0.14 millimicron.

It is important that we have a clear conception of what "wave length" means. According to the wave theory, the wave length of a light-ray is the velocity of light divided by the frequency of vibration. This theory supposes a vibrating body to be in a medium which is thrown into waves by the vibration. These waves all travel with the same velocity, whatever the frequency of vibration. The number of vibrations executed per second is known as the frequency. The waves are equally spaced, and the length of wave will be the length of a series requiring one second to pass any point, divided by the frequency of vibration. The waves of the electromagnetic spectrum have varying physical properties and physiological effects; the visible rays have little if any proved physiologic effect. The infra-red are heat-producing, and the ultra-violet, Grenz, roentgen, and gamma rays produce chemical changes in the body tissues.

In ancient times the sun was associated with the healing art. Apollo, Ra, Baal, and Mithras were sun gods to whom the Greeks, Egyptians, Israelites, Philistines, and Persians prayed for health. While these ancient peoples may have recognized the therapeutic powers of the sun, it is difficult to separate their therapeutics from their religion; hence, therapy from their point of view was, to say the most, quite empirical. Akhnaton, father of Tutankhamen's wife, is given a place in the religious history of the world as being the first individual to attempt to set aside the numerous gods of his subjects, and to substitute for them a single god, called Aton-Ra, meaning "the effulgence derived from Aton." The symbol of this religion was the sun's disc, from which extended numerous rays, each ending in a hand. This symbol was not worshipped *per se*, but the vivifying essence which it typified was.

The Greeks and Romans firmly believed in the hygienic efficacy of the sun's rays, their physicians prescribing them for cure and prevention of all manner of diseases. Herodotus, Pliny, Cicero, and the younger Pliny all pay tribute to the value of light as a medicinal agent, and inform us that solaria were found in the villas of Rome's leading citizens. Hippocrates practised the sun cure in the Greek Archipelago, and Celsus, Galen, and many other leading physi-

cians of their time strongly advocated heliotherapy. Sun worship formed part of the religion of the Aztecs in Mexico.

Modern ideas on radiation are evolved from Newton's discovery, in 1666, of the spectrum of visible light. He failed to detect ultra-violet rays because of a lack of sufficiently delicate apparatus. In 1777, Scheele found that silver chloride was darkened by invisible rays above the violet end of Newton's spectrum. In 1800, Sir William Herschel discovered the infra-red rays. Inglefield, in 1803, suggested that an extension similar to that of the infra-red region might exist at the violet end of the spectrum.

Ritter and Wolloston, in 1801, were the first to demonstrate that the silver chloride in the photographic plate was readily darkened by radiations above the violet end of the spectrum. The honor of the discovery of ultra-violet rays, therefore, belongs to Ritter and Wolloston.

In 1868, James Clerk Maxwell enunciated his electromagnetic theory. Balfour Stewart's discovery that the relation between the powers of emission and of absorption for rays of the same wave length is constant for all bodies at the same temperature, and observations of Fraunhofer's lines elicited knowledge, all of which led to a more accurate idea of the nature of sunlight, and laid the foundation of modern spectroscopy.

In 1868, Ångstrom mapped out the wave lengths of the visible solar spectrum, his unit having since been adopted as the standard of measurement for wave lengths, but it was not until years afterward that serious scientific study was directed to biological effects of light, and precise rays of the spectrum involved in these therapeutics. Scientific interest in this form of therapy dates from 1774, when Faure published a treatise in which he proposed that open sores on the leg should be exposed to the sun's rays. In 1776, two of Faure's followers, Le Peyre and Le Conte, claimed to have cured callous ulcers and slow healing wounds by exposing them to solar rays concentrated through lenses. In 1802, Sir Humphry Davy introduced the electric arc, and, in 1808, exhibited it before the Royal Institute. This discovery made artificial ultra-violet radiotherapy possible.

In 1815, the first comprehensive work on heliotherapy was published by Loebel, of Jena, who devised an apparatus called helio-thermos, a type of box bath quite similar to the modern radiant heat bath, and enumerated the various ailments in which sunlight treatment had proved of value, also others wherein it was contra-indicated. In 1835, Rosenbaum published a work in which he advocated the use of heliotherapy in scrofulous and rickety children. In 1845, Bonnet, of Lyons, extolled heliotherapy in chronic arthritis, especially tuberculous arthritis, by both local and general irradiation. In 1855, Arnold Rikli established an institution for sun bathing in Switzerland, the patients lying naked on couches in special galleries protected from the wind, their head and eyes being shielded.

In 1865, Lister's application to surgery of Pasteur's discovery of micro-organisms as the cause of disease, paved the way to modern ultra-violet treatment.

In 1877, Downes and Blunt, by irradiating two series of culture tubes, one set being surrounded by lead foil which absorbed visible and ultra-violet rays but not heat rays, and the other set being left uncovered, destroyed organisms in the latter, thus proving that chemical rays in sunlight (not heat rays) produce the bactericidal action.

In 1889, Widmark demonstrated that erythema and pigmentation are caused by ultra-violet rays present in sunlight. To Niels Finsen is due primarily the credit for the modern development of ultra-violet radiotherapy. In his initial experiments, in 1892, he discovered how pigmentation protects the skin against excessive radiation. In 1893, Finsen developed his well-known red room treatment for smallpox, and one year later published his first article on the subject, the method therein described having been most successfully employed by Lindholm and Svendson, in Norway.

Finsen devoted particular attention to the study of the injurious effects of the chemical rays as the basis of actinotherapy, referring in this connection to certain diseases which might be aggravated or even caused by exposure to these rays (*i.e.*, blue and violet rays and ultra-violet rays of sunlight). He likewise pointed to the fact that no living tissue absorbs so much light as the blood. He next observed the effect of sunlight on the fetuses of frogs and salamanders, concluding that light, principally violet rays, provoked movements in the fetuses; later, he conducted experiments involving phototropism in various insects.

In 1899, Finsen advocated the use of light-baths, as distinguished from radiant-heat baths, whereby the entire body could be irradiated with so-called cold chemical rays, recommending them especially in the treatment of tuberculosis. Two years previously, he had published his most widely known work on the treatment of lupus vulgaris by concentrated chemical rays. Finding sunlight undependable as a source of ultra-violet, he resorted to the electric arc, designing a lamp wherein the divergent rays were rendered parallel by a system of lenses. He proved by experiments that concentrated rays had the greatest bactericidal effect, discovering that concentrated sunlight killed bacteria fifteen times faster than direct radiation, and that concentrated arc light was even stronger. He demonstrated that the blood markedly absorbs ultra-violet rays, and limits their penetration of the tissues.

In 1902, Bernhard began a revival of heliotherapy in Switzerland. He irradiated ulcers of all types, but especially those of tuberculous origin, and closed cases of non-pulmonary tuberculosis. Rollier, in 1904, encouraged by the success of Bernhard, established a clinic at Leysin solely for heliotherapy of non-pulmonary tuberculosis.

Kromayer, in 1906, designed his water-cooled quartz mercury lamp, still unsurpassed for local ultra-violet treatment when installation of the Finsen lamp is impossible.

In 1908, Nagelschmidt designed his air-cooled quartz mercury vapor lamp for general irradiation. This was the first ultra-violet lamp for treatment of the entire body, notwithstanding Finsen's advocacy of general light-baths ten years previously.

In 1908, Sir Henry Gauvain introduced heliotherapy into England. In 1913, Reyn instituted general carbon-arc-bath treatment in Copenhagen. In 1918, Huldschinsky first used the quartz mercury vapor lamp for the cure of rickets, though Palm, an English physician, in 1890, advocated sun baths as a preventive and cure for this and other diseases.

In 1925, Rosenheim, Webster, Hess, and Windhaus, in different countries, discovered independently that it is possible to produce vitamin D artificially by exposing a non-saponifiable fat called ergosterol to ultra-violet radiation. Since ergosterol is present in human skin, production of vitamin D in the tissue of the epidermis constitutes an important action of ultra-violet irradiation.

Mayer states that ultra-violet radiation is of value in calcium deficiency diseases, tetany, rickets, osteomalacia, and in overcoming the demineralization of the pregnant and nursing mother, also, as an aid in superficial ulcerations, certain skin diseases and extrapulmonary forms of tuberculosis, lupus vulgaris, and scrofuloderma, and that it possesses occasional usefulness in acne vulgaris, psoriasis, indolent ulcers, pityriasis rosea, adenoma sebaceum, and erythema induratum. Its value is questionable in the treatment of boils and generalized staphylococcus infections of the skin, and alopecia.

In tuberculosis, light comprises only one of the important adjuvants in treatment. Employing light as an aid only, the most favorable response to solar exposures has been shown in the so-called "pretuberculosis" of children, and in tuberculosis of the lymph nodes including the hilus, pleura, bones and joints, peritoneum and intestine. Less favorable, yet often good, results are obtained in genito-urinary, laryngeal, ocular, aural, and cutaneous tuberculosis. Pulmonary tuberculosis is not considered an indication for light therapy. Post-

operative sinuses after nephrectomy are especially responsive to ultra-violet irradiation.

As a closing word with respect to ultra-violet radiation, it may be said that reliance on any source of light as an important aid in pulmonary tuberculosis is not to be encouraged, as harm may be done by the injudicious and uninformed use of light. While ultra-violet radiation is valuable in a limited number of diseases, it should not be prescribed generally by those unfamiliar with its contra-indications and details of its application.

Grenz rays are soft roentgen rays having wave lengths of from 1 to 3 Ångstrom units, and are produced in lithium glass x-ray tubes with voltages of from 4 to 10 kilovolts. The exact position of the Grenz-ray spectrum in the general spectrum of radiation has been previously mentioned. The apparatus for the production of Grenz rays may be described briefly as a high tension transformer with closed iron core. The high tension is regulated by both auto-transformer- and resistance-control. One of the high tension lines is grounded. Kilovoltmeter and milliameter are built into the switch board. Some observers believe that an undistorted voltage curve may be obtained by the use of valve tubes to rectify the current of the transformer.

With regard to the absorption of Grenz rays, it may be stated that while 90 per cent of roentgen-ray energy passes through the skin, only 12 per cent of Grenz rays reach the subcutaneous tissues, 88 per cent being absorbed by the skin. Further biologic considerations and technic of application are not within the scope of this paper.

The discovery of roentgen rays was not an accident, as has been stated in the lay press and pseudo-scientific papers, but was the culmination of the work of many investigators over a long period of time.

Thales, of Miletus, in 600 B.C., and Theophrastus, in 321 B.C., discovered independently the first electrical phenomenon, which was friction causing amber to attract light objects. These observations were entirely forgotten, but were rediscovered in the Middle Ages, and the real birth of our present-day knowledge of electricity and magnetism dates from this rediscovery.

William Gilbert, 1540–1603, devoted many hours to research. In 1600, after eighteen years of hard work, he published his famous treatise under the title of "The Magnetic Bodies, that Great Magnet, the Earth; the New Physiology, Set Forth by Many Arguments and Experiments." This work was recognized throughout the civilized world as authoritative information on magnetism and "electrical" observations, and formed the real scientific foundation of subsequent investigations of electricity and magnetism. Gilbert discovered magnetic induction, magnetic conductivity, and terrestrial magnetism, that is, that the earth itself is a great magnet. He rediscovered frictional electricity and the electrical properties of amber, and in addition he found a series of other materials such as sulphur, glass, resin, sealing wax and many crystals, which, when rubbed, also possess "electrical properties." Nicolaus Cabaeus, 1585–1650, a Ferrara Jesuit, was the first to record the phenomenon of electrical repulsion, in 1629.

Evangelista Torrecelli, 1608–1647, was among those who first attempted to create an empty space, that is, to investigate the basic principles of removing air from an air-tight vessel. Thus he contributed to the development of the evacuated glass tube so necessary in x-radiation therapy.

Otto von Guericke, 1602–1686, invented an air-pump, and, in 1654, demonstrated to Emperor Ferdinand III the Magdeburg hemispheres. At that time he illustrated the outside air pressure on the evacuated hemispheres, which were about twenty inches in diameter, by showing that two teams of eight horses were unable to pull the hemispheres apart; but, as soon as air was admitted to the evacuated space, the hemispheres fell apart. Von Guericke also constructed an electrical machine which consisted of a rotating sulphur sphere against which he pressed one of his hands, yielding static electricity of a potential high

enough to produce phosphorescent light and brush-discharges or even small sparks.

Robert Boyle, 1627–1691, improved von Guericke's air-pump by adding the pinion movement to it. This improved pump was followed later by still more improved types by Hooke and Papin, and by Boyle himself. In 1667, he made the important observation that the electrostatic effects of rubbed amber and magnetic effects were also efficacious in a vacuum.

Isaac Newton, 1643–1727, built, in 1675, an electrical machine with a rotating glass sphere, with which he made a large series of important experiments.

Francis Hauksbee, the elder, who died in 1713, built a static machine using wool which rubbed against glass, and later, leather rubbing against amber. He also investigated an effect known as "mercurial phosphorus," consisting of a flickering luminescence produced by shaking mercury in an evacuated tube. Proceeding a step farther in studying these light phenomena, he placed the electrical machine in a bottle which could be evacuated. With this apparatus he produced brush-discharges, and therewith many effects of "electrical light, and also larger sparks than had ever before been seen in a laboratory."

Stephen Gray, 1696–1736, in 1729 made the important discovery that electricity could be conducted from one body to another through metallic wires.

Charles Francis de Cisternay du Fay, 1699–1739, in 1734 made the discovery of two kinds of electricity, positive and negative, which he called "vitreous" and "resinous."

Jean Antoine Nollet, 1700–1770, simplified the Hauksbee apparatus by placing the source of the high tension outside the evacuated vessel, and led the high tension to the glass vessel by means of iron chains. The glass vessel was egg-shaped, and it was therefore called the "electrical egg." This "electrical egg" was undoubtedly the forerunner of the Hittorf-Crookes tubes, and also of the roentgen-ray tube.

William Morgan (died 1785) perhaps was the first one actually to produce x-rays. In performing experiments with vacuum tubes and electric currents, he produced a vacuum so high that he did not succeed in obtaining a discharge of the high tension through the tube. At this time, in all probability, x-rays were produced.

Others who made contributions to the knowledge of electricity were Benjamin Franklin (1706–1790), C. A. Hansen, of Leipzig (1693–1743), G. M. Bose, of Wittenberg (1710–1761), F. H. Winkler, of Leipzig (1703–1770), P. Gordon (1712–1751), B. Wilson (1712–1788), Bennett, Erasmus Darwin, and G. C. Bohnenberger, who wrote six extensive treatises on the history of electrical machines, and notably on his own constructions. LeRoi and Chevalier, of France, were probably the first to measure electricity quantitatively by means of an electrometer. John Canton (1718–1772), in England, also constructed a measuring device of pith-balls. J. K. Wilcke (1732–1796), a Swede, and Beccaria, of Italy, also experimented with electrostatic phenomena. Others who in the eighteenth century discovered a new kind of electricity in contradistinction to frictional electricity were, notably, the Italians, Luigi Galvani (1737–1798) and Alessandro Volta (1745–1827), and the Frenchman, Coulomb.

Hans Christian Oerstedt (1777–1851), a Dane, conducted most important experiments on the interrelationship of electrical and magnetic phenomena. His most famous discovery was the deviation of a magnetic needle by a conductor through which a current was flowing.

Andre Marie Ampere (1775–1836) elaborated and enlarged upon the work of Oerstedt; and Arago and H. Davy observed that a connecting wire of a voltaic battery could magnetize iron.

G. S. Ohm (1787–1854) contributed Ohm's law, a mathematical formula stating the relationship of current, electromotive force, and resistance.

Michael Faraday (1791–1867), a contemporary of Ohm, made the first induction ring, which led directly to the transformer which to-day produces the high

voltage currents necessary in roentgenology.

Joseph Henry (1797–1878) made an electromagnet capable of sustaining fifty times its own weight. He developed the principle of the first electromagnetic telegraph and of the dynamo, and finally made a thorough study of producing induced currents. Henry was the first secretary and director of the Smithsonian Institution, and delayed publication of his works is responsible for his not sharing equal fame with Faraday.

Wilhelm Weber (1804–1890), of Germany, and H. F. E. Lenz (1804–1865), of Rumania, enlarged upon some of Faraday's observations. Weber determined the absolute measure of electromagnetic force, and Lenz studied quantitatively the relation between electromagnetic induction and the mechanical action of electric currents.

G. Belli, in 1831, G. F. Varley, August Loepler (1836–1912), W. T. B. Holtz (1836–1913), James Wimshurst, and R. Voss all made notable contributions to the improvement of electrostatic machines. After the discovery of roentgen rays, these machines were frequently used to produce the high potential necessary to excite the x-ray tube.

R. Kohlrausch (1809–1858) and Sir William Thomson (Lord Kelvin, 1824–1907) studied and constructed instruments to measure potential and current.

In 1843, a Frenchman, Abria, observed the striated effect in vacuum tubes and studied the influence of polarity.

Grove, Quet, Sequin, and J. P. Gossiot contributed many important observations on evacuated tubes.

The first induction coils, electro-induction principle, were built by W. Sturgeon, in 1836, and by C. G. Page, in 1838.

C. E. Neef, J. P. Wagner, and J. W. Gouley improved the apparatus by adding the mechanical interrupter, and, in 1853, H. I. Fizeau added the condenser. H. D. Ruhmkorff constructed the best induction coils of the time, hence the frequent use of his name in this connection.

Herman Spreugel developed the mercury air-pump, in 1865. A relatively high vacuum could be produced very rapidly, and thereafter better tubes resulted.

James Clerk Maxwell (1831–1879) successfully translated Faraday's work into the language of mathematics. He reported his work in the book, "A Dynamical Theory of the Electromagnetic Field," 1863, and in a paper entitled "A Treatise on Electricity and Magnetism," 1873.

Heinrich Rudolph Hertz (1857–1894) experimentally substantiated Maxwell's creative theory, twenty years after Maxwell's death. Hertz also continued the researches of his predecessors on the electric discharge in rarefied gas. The latter work was made possible by the development of Geissler tubes.

H. Geissler (1815–1879) constructed glass tubes capable of being exhausted to a fairly high degree of vacuum, or filled with different gases under pressure. Experimenting with electrical discharges in these gas-filled tubes, he noted that wonderful color effects were displayed.

Julius Plucker (1801–1868) encouraged Geissler in his work, and together with Geissler investigated the spectrum obtained from gases enclosed in Geissler tubes.

Wilhelm Hittorf (1824–1914) studied the influence of a magnet on the radiant emanations from the cathode of a Geissler tube; also, he made tubes having a much higher vacuum than Geissler tubes. One of Hittorf's most important observations was that the light from the negative electrode (cathode) of the vacuum tube leaves the electrode in straight lines, produces heat, and causes fluorescence on the glass where it impinges. In 1869, he discovered that when solid matter is brought into the path of this light, a shadow of it is cast on the glass walls of the tube. It is within the bounds of probability that Hittorf produced x-rays at this time (1869).

William Crookes (1832–1919) substituted and enlarged upon Hittorf's work, his most important studies being on electrical discharges through rarefied gases, and was given credit for observations made

many years before by the modest Hittorf.

H. von Helmholtz (1821–1894) presented a theory of electromagnetic dispersion based on Maxwell's equations. He described the phenomena of deflection, diffraction, and penetration, and even predicted theoretically Roentgen's great discovery.

Phillip Lenard (1862–) demonstrated the transmission of cathode rays through metals, and found that they could be propagated through air after being produced, and also through various gases. He noted too that the effects of cathode rays could be studied with fluorescing substances such as phosphates and ketones. In addition, he found that cathode radiation darkened photographic plates.

Wilhelm Conrad Roentgen (1845–1923) started his practical experiments with high tension apparatus and electrical discharges through rarified gases, in October, 1895. Soon thereafter he discovered the x-rays, on Nov. 8, 1895. This discovery was not an accidental happening, but was the brilliant climax to the studious application and previous accomplishments of many other scientists—Lenard, von Helmholtz, Hertz, Hittorf, Crookes, and before them, Maxwell, Faraday, Ohm, Ampere, and then Galvani, Volta, Franklin, Henry, and others.

During the early days following Roentgen's discovery, curiosity concerning possible therapeutic effects was rife, and some of the bolder spirits attempted to treat disease empirically. E. H. Grubbe, of Chicago, applied roentgen rays to a patient with carcinoma of the breast, Jan. 29, 1896, and the following day to a case of lupus vulgaris. Grubbe at this time was suffering with a dermatitis which had apparently been caused by exposure to the rays while experimenting with vacuum tubes, of which he was a manufacturer at the time Roentgen made his discovery. Not being a physician, Grubbe did not publish a report of the cases treated empirically, at the request of Dr. J. E. Gillman, who attended his dermatitis.

A Dr. Voight, of Hamburg, Germany, presented a case of carcinoma of the nasopharynx treated by x-rays, to the Society of Physicians of Hamburg, on Feb. 3, 1896. The patient was relieved of pain. In the wave of enthusiasm that followed this new discovery, deleterious effects were not considered. All its effects were thought to be beneficial only. A false sense of security was built up, and many and various were the causes given for the frequently occurring dermatitis. Among the first to recognize the true cause of the skin changes were Elihu Thompson, Francis H. Williams, and William Rollins, all of Boston.

The epilating effect of x-rays was another manifestation noted. This phenomenon, and not the erythema, was the real clue to the establishment of roentgen therapy. Those first reporting their success, and generally given credit by authorities for priority in treating various conditions are as follows: Hypertrichosis, Freund, 1897; lupus, Kummel and Schiff, 1897; lupus erythematosis, Schiff, 1898; psoriasis, Ziemssen, 1898; sycosis and favus, Freund, 1898; nevus vascularis, Jutossy, 1898; chronic eczema, Hahn, 1898; epithelioma, Sjogren and Stenbeck, 1899; alopecia areata, Kienbock, 1900; superficial sarcoma, Ricketts, 1900; mycosis fungoides, Scholtz, 1902; leukemia and lymphadenomas, Pusey and Senn, 1903.

In the early days of roentgenology, mechanical difficulties were many and various, and the means of accurately determining dosage were not known. At this stage, the only way of estimating the intensity of output or dosage was based upon fluoroscopic or photographic effects. Freund said that he used an intensity "which would take a roentgen photograph of a man's hand at 15 cm. distance in one minute." Fluoroscopes were used to determine the hardness or softness of a tube by the sharpness of the image of the hand. Thus many burns resulted until the danger was recognized—in many instances, too late.

Ennion G. Williams, in 1903, was the first to call attention to the fact that the tube should be placed at a greater distance from the skin to treat deep-seated lesions.

The first satisfactory method of measuring the output was the chromoradiometer suggested by Guido Holzknecht. L. Benoist described a penetrometer which was the first device to measure the quality of radiation. The developments in the field of measurement may be summarized as follows: (1) measurement of electric current; (2) penetration method of Benoist; (3) skiometers and penetrometers using other metals; (4) physicochemical methods; (5) ionization methods; (6) photometric methods.

In 1903, H. Albers-Schönberg announced that aspermia could be produced in experimental animals by roentgen rays, and Nicholas Senn really founded deep therapy when he reported favorable results from irradiation of the spleen in leukemia.

In 1904, J. Bergonie and L. Triboudeau gave the complete histologic picture of the changes produced by irradiation upon the rat's testicle, and showed the point of attack of the roentgen rays to be on the embryonic cellular structures.

In 1904, G. Perthes reported an extensive investigation of the rate of absorption of roentgen rays in tissue, and proved that when the rays are filtered through aluminum the skin is protected, and the intensity into the deeper layers is increased. He made the first depth-dose measurements which led to the use of filters in Continental Europe.

George E. Pfahler, in 1906, presented a paper on the subject of filters, and he is responsible for the use of filters in America.

Improvements in equipment were developing during this time, including tubes, but it was late in 1913 when Dr. W. D. Coolidge announced the "hot cathode" tube. This type of tube is now universally used in roentgen therapy.

Cancer of the breast was one of the first malignant diseases to which x-rays were applied for therapeutic purposes. Tuberculosis in its various manifestations was treated in the first years, and as early as 1904, 518 cases of surgical tuberculosis were found in the literature, with reported cures of 42 per cent. The treatment of hyperthyroidism began early, the subject being discussed by Carl Beck in 1900. Cervical adenopathy, prostatic hypertrophy, carcinoma of the bladder, carcinoma of the stomach, and trigeminal neuralgia were treated by various observers, who reported successful results. The analgesic effects from the rays were noted shortly after their discovery, and attempts were made to treat epilepsy, with reports of satisfactory improvement and diminution of the number of attacks.

Syringomyelia and other lesions of the nervous system were treated before the first years of the twentieth century. Among those discussed in the literature are neuralgia, sciatica, herpes zoster, and general and progressive paralytic diseases. Intracranial tumors were treated by Henri Béclère, of Paris, who, in 1909, reported results in a series of about forty cases of tumors of the hypophysis.

In 1915, Kupferle and V. Slizy, in Germany, made a more extensive study, and Nordenlabt reported on other types of intracranial neoplasms. By 1910, neoplasms in all parts of the body were being treated, which with improvement of technical procedures gave improved results.

Attempts to improve on the methods of treating gynecologic conditions brought about decided advances and improvements in roentgen therapy. The first condition of the female pelvis to be treated was uterine fibromyoma, therapy being based on the sterilizing influence of the rays on the ovaries.

The treatment of carcinoma of the cervix began about 1902, and applications were made by directing the rays through specula, and tubes which could be placed directly against the cervix were also designed for this special purpose. Dr. Eugene W. Caldwell, in whose honor the annual Caldwell Lecture of the American Roentgen Ray Society is given, designed some especially good tubes for cervical application. This effort to treat gynecologic conditions led to our modern cross-fire technic, which was first adapted to the treatment of pelvic conditions.

During the second decade of the twentieth century, rapid strides were made in apparatus, tubes, and the correlation of physical conditions with roentgen therapy. Homogeneous radiation technic was born. This led to the multiple cross-fire method and fundamental researches in pure physics on the quality (wave length) and quantity (intensity) of roentgen rays. These researches were the basis of scientific medical radiology. The following physicists made notable contributions to this phase of roentgen therapy: from Germany, M. von Lane, W. Friedrich, and P. Kuipping; from England, W. H. and W. L. Bragg, C. G. Barkla, and C. A. Sadler; from Sweden, M. Siegbohn; from other points, M. de Broglie, H. Seeman, W. Duane, F. Hunt, T. Christen, B. Szilard, O. Glasser, and many others.

In 1928, a unit for measuring the quantity of radiation was finally established. This was named the "roentgen," to be designated by the letter "r." The International Congress of Radiology, held in Stockholm in 1928, officially adopted this unit by international agreement, and a problem previously thought incapable of solution was solved. Modern methods of treatment with radium and roentgen rays are now practically an exact science. The depth dose at any level in the body may be accurately determined, as can the total dosage delivered to any organ. This is accomplished by means of charts showing the percentage rate of absorption. As an example of this, it may be stated that at a depth of 20 cm., only 10 per cent of effective radiation is available, compared with 100 per cent at the skin surface.

At the present time, the whole procedure of applying roentgen therapy to deeply situated regions of the body is based upon the effort to direct highly penetrating rays produced by high potentials (200 to 1000 kv.) and thick metallic filters (0.5 mm. to 5.0 mm. of copper), with the source of the rays at comparatively long distances (40 to 75 cm.).

The most important biologic indication of dosage has been the human skin, because its reaction and the possibility of damage to it limit the irradiation which can be applied during the treatment. It is axiomatic that the shorter the rays, the deeper the penetration; the more scattered radiation which affects the skin, is proportionately less than the direct radiation from the tube.

In the beginning of roentgen therapy, the method of applying roentgen rays had been based upon "divided doses," frequently repeated with one field, directly over the diseased area. It was found, in 1900, by treating some conditions, that it was desirable to create a definite dermatitis or inflammatory reaction in the diseased area to obtain the best results, especially for superficial malignant processes. Many controversies arose concerning the necessity or advisability of producing an inflammatory reaction. In contrast, modern roentgenologists are unanimous in the opinion that it is advisable to administer the largest possible dose of radiation to most neoplastic diseases. The method may be the massive dose technic, in which the largest possible dose is administered in the shortest possible time. The saturation method of Pfahler, in which an attempt is made to keep the diseased tissues saturated with radiation over a long period of time during cell division, by doses carefully calculated as to *per diem* loss and normal tissue tolerance, may be employed. The Coutard method, especially applicable for treatment of malignant disease of the upper respiratory tract, is conducted by utilizing very heavy filtration, and giving two doses each day over a very long period of time (25 to 35 days). Another procedure has been tried for certain diseases (Hodgkin's disease and leukemic disease). This is the application of total prolonged radiation, the body of the patient being subjected to very small intensity, day and night.

At the present time, potentials as high as a million volts are available, and the physical and biologic aspects of the problems are being studied with the hope that the clinical effects in roentgen therapy will bring about better results.

Radium was discovered by Pierre and

Marie Curie, in 1898. This discovery was the result of a false conclusion drawn by Becquerel, that the association between fluorescence and roentgen rays existed also in the case of substances which fluoresced under the action of sunlight. This deduction, while not scientifically true, led to the discovery of polonium (named by Marie Curie for her native Poland) and radium. Investigation of this new element revealed the fact that three distinct types of radiation are emitted: Alpha rays, which are not actually rays, but helium nuclei travelling at speeds of 9,000 to 20,000 miles per second. These rays, or, more accurately, alpha particles can be stopped by substances no more dense than a single sheet of paper. Beta rays are also not rays, but particles. They are the same type as cathode rays, and are, therefore, negative electrons travelling at high speeds which may vary considerably, some of them reaching almost the speed of light, 186,000 miles per second. They are much lighter than alpha particles, but travel much faster, and they can penetrate much farther into matter; the swifter ones can traverse a centimeter of tissue. They are all stopped by two millimeters of brass, or one-half millimeter of gold. The gamma rays are truly rays, in the same sense as roentgen rays or visible light. They are of very short wave length, and consequently of great penetrating power. The hardest gamma rays can pass through 25 cm. of lead (approximately 10 inches).

Radium has an atomic weight of 226, and exhibits the power of spontaneous disintegration, as do all elements with atomic weights higher than 208. About forty radio-active elements are known. These may be classified in three series: the uranium, thorium, and actinium series. Radium being a self-destructive element, the question naturally arises as to how long the radium atom may exist as such. It may be stated that it may last only a few seconds, or it may endure for thousands of years. Suitable experiments demonstrate what may be called the average life of the atoms of any element. The average life of the radium atoms has been found to be 2,400 years. In any given amount of pure radium, the amount of radium gradually decreases. In 1,690 years, one-half of it will be disintegrated; the half that is left will behave in the same manner, and so on to infinity.

The disintegration of radium gives rise to a gas, radon, which has a half-value period of 3.85 days. It is chemically inert, but nevertheless very radio-active. Radon is collected by comparatively simple apparatus, sealed in glass, gold, or platinum seeds, and used in the war against disease, particularly malignancy. In the treatment of malignancy with radium, the following factors are considered and specified: (*a*) location of tumor; (*b*) size of tumor; (*c*) depth of tumor; (*d*) strength of radio-active source; (*e*) distribution of radio-active source; (*f*) filtration; (*g*) distance of spacing; (*h*) duration of irradiation.

The biologic effects of radium must be understood if it is to be used intelligently and with the maximum beneficial effect. The first effect is an alteration in the morphology and physiology of the cell, which may occur after a short or a long latent period, depending on the quantity and quality of the radiation. The rate of cell division is altered. Those cells which are in mitosis at the time of exposure complete that division, and then remain quiescent for some time, while those not actually dividing are prevented from so doing. Thus, practically all mitosis comes temporarily to an end soon after exposure.

The primary biologic effects of radiation may be summarized as follows: changes in the hydrogen-ion concentration of protoplasm, in the permeability of the cell membrane, in the viscosity, and in the respiratory rate. The morphologic effects are secondary.

At the present time, there are over four hundred different diseases which are benefited by roentgen therapy, and the rationale of modern roentgen and radium therapeutics is based on knowledge of the effects of the rays upon the associated morbid anatomic changes which are the result of

disease processes. From clinical observation and biologic researches it is known that the different types of cells or tissues of which the living organism is composed vary in their sensitiveness to radiation in the following order:

(1) Primitive blood cells.
(2) Germinal cells of ovary and testicle.
(3) Blood-forming tissues invading the cells of the red bone marrow, lymphatic system, and spleen.
(4) Thymus, pituitary, adrenals, and thyroid.
(5) The skin and its glands and hair follicles.
(6) The liver, intestines, pancreas, kidney, and uterus.
(7) Connective tissue, muscles, fascia, tendons, cartilage, bone, fat, and nerve cells.

In conclusion, it may not be inappropriate to point out that the roentgen therapist is primarily a physician who has set for himself a task of treating conditions and diseases with which other skilled medical artisans are powerless to cope. Roentgen therapy has been directed toward ameliorating unmedicable ailments; therefore, the roentgen therapist must necessarily meet with many failures. Yet, his successes have encouraged him to tireless effort, which he will continue until a better method has been conceived, yielding still greater accomplishments.

The following is a list of diseases and conditions amenable to radiation therapy. A key to the symbols will be found at the end of the list (page 98).

1. Acne vulgaris X-R-G-U (f)
2. Acne rosacea X-R-G (h)
3. Acne varioliformis X-R (f)
4. Acne keloid X-G-R
5. Acanthosis nigricans X (c)
6. Actinomycosis X-R (d)
7. Angina pectoris X-U
8. Arthritis deformans X
9. Arthritis, gonorrheal X
10. Asthma, bronchial X-U
11. Angina, agranulocytic X
12. Adenitis X
13. Adenoma sebaceum U
14. Alopecia U?
15. Amenorrhea X-U
16. Abscess, ischiorectal U
17. Abscess, perineal U
18. Achondroplasia U
19. Anemia, second. pernic. U
20. Asthenia, suprarenal U
21. Angioma R-G (m)
22. Angulus infectiosis G
23. Addison's disease X (c)
24. Angioma cavernosum R (d) (m)
25. Angioma, senile R (d) (m)
26. Blastomycosis X-R (d)
27. Bowen's precancerous dermatosis X
28. Bromidrosis X (a)
29. Blepharitis U
30. Bronchitis U
31. Bronchiectasis U
32. Bronchopneumonia U
33. Bubo X
34. Carbuncle X-R (d)
35. Carcinoma, mammary X-R
36. Carcinoma, cervix R-X
37. Carcinoma, tongue R-X
38. Carcinoma, larynx X-R
39. Carcinoma, lung X
40. Carcinoma, stomach X
41. Carcinoma, colon X
42. Cerebral tumors X
43. Chilblains X
44. Corns X (f) (m)
45. Condyloma acumina X
46. Calcium deficiency tetany U
47. Calcium deficiency rickets U
48. Calcium deficiency osteomalacia U
49. Calcium deficiency pregnancy U
50. Constipation U
51. Cervical erosion U
52. Cirrhosis of the liver U
53. Cholecystitis U
54. Convulsions, reflex U
55. Cystitis (T.B.) U
56. Cardiac, functional U
57. Chorea U
58. Chickenpox U
59. Choroiditis U
60. Conjunctivitis U
61. Coryza U
62. Chorioretinitis U
63. Callositas X-G (i)
64. Cheilitis, exfoliativa X (f)
65. Cheilitis, glandularis X (f)
66. Chromidrosis X (a)
67. Cornu X-G
68. Dermatitis exfoliativa X (j)
69. Dermatitis infectiosa eczematoides X
70. Dermatitis papillaris capillitii X (a)
71. Dermatophytæ X
72. Dermatophytosis X-G
73. Diabetes mellitus U
74. Dyspepsia U
75. Diarrhea U
76. Dysmenorrhea U
77. Dementia præcox U
78. Diphtheria U
79. Diphtheria carriers U
80. Dacryocystitis U
81. Dermatitis herpetiformis G
82. Eczema X-G (j)
83. Erysipelas X (l)

84. Epithelioma R-X-G (e) (i) (l)
85. Erythroderma G
86. Erythema induratum X-U-R (e)
87. Empyema U
88. Epididymitis U
89. Encephalitis (Parkinson) U
90. Epistaxis U
91. Eczema seborrhoicum X-G
92. Eczema venenatum X-G
93. Eczema hemostaticum X
94. Favus X (a)
95. Folliculitis decalvans X (l)
96. Furunculosis X-G (g) U?
97. Fibrositis U
98. Fistula U
99. Facial palsy U
100. Fissures G
101. Fibroids, uterine X
102. Gout U
103. Gastric ulcer U
104. Gonorrhea U?
105. Gangrene, senile U
106. Gingivitis U
107. Granuloma annulare X-R (d)
108. Granuloma fungoides X
109. Granuloma pyogenicum X (l)
110. Granulosis, vulva, nasi X (c)
111. Hodgkin's disease X (b)
112. Hydrocystoma X
113. Hyperidrosis X (a)
114. Hypertrichosis X Special (i)
115. Hemorrhoids U
116. Hysteria U
117. Hemophilia U
118. Hypothyroidism U
119. Hypoparathyroidism U
120. Hyperpiesis U
121. Hay fever U
122. Hyperkeratosis—Warts X-G-R
123. Hyperkeratosis—Corns X-G
124. Hyperkeratosis—Cornu cutaneum X-G
125. Hemangioma R-X
126. Hyperthyroidism X
127. Iritis U
128. Iridocyclitis U
129. Indolent ulcers U
130. Incontinence of urine U
131. Infantilism U
132. Impotence U
133. Intertrigo X
134. Jaundice U
135. Keloid X (a)—G
136. Keratosis arsenicalis X-R (e) (m)
137. Keratosis follicularis X (e) (m)
138. Keratosis senilis X-R (e) (m)
139. Keratosis vulvæ X (l)
140. Keratitis U
141. Kaposi's disease G
142. Keratosis pilaris G
143. Lichen planus G-X (j)
144. Lupus erythematosus G-X (k) (m)
145. Lupus vulgaris G-X-U (e)
146. Leukemia X
147. Lymphosarcoma X
148. Lumbago U
149. Laryngismus stridulus U
150. Leukorrhea U
151. Locomotor ataxia U
152. Laryngitis U
153. Leukemia cutis X (b)
154. Leukoplakia R (m)
155. Lichen nitidus X (l)
156. Lichen scrofulosus X (l)
157. Lichenification X
158. Lingua geographica X (c)
159. Lupus miliaris disseminatus X-R (d)
160. Lymphangioma circumscriptum R (m)
161. Lymphangitis X (l)
162. Lymphogranulomatosis cutis X (b)
163. Molluscum contagiosum X (l)
164. Myositis U
165. Marasmus U
166. Menopause U-X
167. Menorrhagia U-X
168. Metrorrhagia U-X
169. Measles U
170. Mumps U
171. Mastitis X
172. Mixed tumors, parotid X
173. Multiple myeloma X
174. Mycosis fungoides X (b)
175. Melanosarcoma G
176. Nevus flammeus G
177. Nephritis, pain U
178. Nephritis, headache U
179. Nephritis, insomnia U
180. Nephritis, diuresis U
181. Neuritis U
182. Neuralgia U
183. Neurasthenia U
184. Neurodermatitis X
185. Neurodermatitis, disseminate X
186. Nevus pilosis X Special (i)
187. Onychomycosis X (g)
188. Obesity U
189. Osteoporosis U
190. Osteomalacia U
191. Osteogenesis imperfecta U
192. Osteomyelitis U-X
193. Orchitis U
194. Ozena U
195. Otitis media U
196. Osteitis fibrosa cystica X
197. Parapsoriasis G-X (e)
198. Psoriasis G-X-U (j)
199. Paronychia G-X (j)
200. Pruritus ani G-X (b)
201. Pruritus vulvæ G-X (b)
202. Paget's disease X-U (i)
203. Pain X
204. Parathyroid dyscrasias X
205. Paratitis X
206. Pertussis X
207. Phlebitis U
208. Prostatic hypertrophy X-U
209. Purpura hemorrhagica X-U
210. Pityriasis rosea X-U (j)
211. Pneumonia U
212. Pleurisy U
213. Pleurodynia U
214. Pyelitis U
215. Paralysis agitans U
216. Poliomyelitis U
217. Pharyngitis U
218. Perichondritis U
219. Pyorrhea alveolaris U

220. Perlèche X (c)
221. Pernio X (l)
222. Pompholyx X (j)
223. Prurigo X (k)
224. Rhinophyma X (i)
225. Rhinoscleroma X (a)
226. Rheumatoid arthritis U
227. Raynaud's disease U
228. Rubella U
229. Rhinitis U
230. Ringworm X
231. Sarcoid G-X (e)
232. Sycosis barbæ G-X
233. Sinuses and fistulæ X
234. Splenomegaly X
235. Superficial ulcerations U
236. Spasmophilia U
237. Scrofuloderma U-X-R (d)
238. Staphylococcus infections U
239. Sterility U
240. Sciatica U
241. Scarlet fever U
242. Sinusitis X-U
243. Suppurative mastoiditis U
244. Sarcoma X (b) (i)
245. Sebaceous cyst X
246. Seborrhea oleosa X (j)
247. Sycosis, lupoid X-R (d)
248. Sycosis vulgaris X-R (d)
249. Synovial lesions of skin X-R (d)
250. Syringoma X (l)
251. Tuberculide X (k)
252. Tuberculosis orificialis X (e)
253. Tuberculosis verrucosa cutis X (e)
254. Torticollis U
255. Trachoma U
256. Tonsillitis U
257. Tuberculosis, pulm., adjuvant only (?)
258. Tuberculosis, pre-, of children U
259. Tuberculosis, lymph nodes U
260. Tuberculosis, hilus glands U
261. Tuberculosis, bone U
262. Tuberculosis, pleura U
263. Tuberculosis, joints U
264. Tuberculosis, peritoneum U-X
265. Tuberculosis, intestinal U
266. Tuberculosis, genito-urinary U
267. Tuberculosis, laryngeal U
268. Tuberculosis, ocular U
269. Thymic enlargement X
270. Tonsillar hypertrophy
271. Tinea capitis G
272. Tinea corporis G
273. Unresolved pneumonia X
274. Urethritis U
275. Verruca plantaris X-R (d)
276. Verruca seborrheica R
277. Verruca vulgaris
278. Verruca X
279. Vaginitis U
280. Vitamin D production U
281. Whooping cough U
282. Xanthomatosis X-G

KEY TO SYMBOLS

X, Roentgen rays
R, Radium rays
G, Grenz rays
U, Ultra-violet rays

Do not overlook usual medical or surgical procedures.

1. X (a), Roentgen rays specific
2. X (b), Roentgen rays only useful treatment
3. X (c), Roentgen rays occasionally useful
4. X (d), Roentgen rays method of choice
5. X (e), Roentgen rays in combination with medical or/and surgical treatment
6. X (f), Roentgen rays or medical and surgical treatment
7. X (g), Roentgen rays uncertain
8. X (h), Roentgen rays useful after medical or surgical failure
9. X (i), Roentgen rays not method of choice
10. X (j), Roentgen rays annoying symptoms only
11. X (k) r, or roentgen rays temporary value
12. X (l), r or roentgen rays experience not sufficient to evaluate
13. X (m), Roentgen rays less valuable than radium

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CASE REPORTS AND NEW DEVICES

AN UNUSUAL TYPE OF PULMONARY METASTASIS IN HYPERNEPHROMA

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We wish to present the findings in a patient with hypernephroma because the clinical picture and roentgen manifestations of the disease were unique in our experience.

CASE REPORT

The patient, a white male, aged 44 years, was admitted to the hospital on Sept. 13, 1934, complaining of pain in both lumbar regions. The patient's history began three months prior to his admission to the hospital when he first noticed an ache in his left shoulder. The pain, which was dull and constant, was not increased by motion or deep respiration; it persisted in spite of local applications, and within several weeks became much worse, radiating across the back into the right shoulder region. Since that time the pain had gradually descended until at the time of his admission it was localized in both lumbar areas. During the period of his illness, the patient had lost 12 per cent of his normal weight. About two weeks prior to hospitalization, he developed a cough which at first was mucopurulent but later became blood-streaked. The gastro-intestinal history was negative. The genito-urinary history was also negative, except for a moderate oliguria that started two weeks before the patient entered the hospital.

The patient's past medical and family histories were unimportant and threw no light upon his malady.

The physical examination revealed an undernourished, white man of Irish extraction, who was well oriented in all spheres. The skin presented a fine erythematous and diffuse papular eruption. All of the superficial lymph glands were moderately enlarged but their texture seemed normal. The patient's blood pressure in both arms was 124/72. The skeletal system was negative with the exception of a marked degree of clubbing of all the fingers and toes, which the patient insisted had been present for many years. The eyes, ears, nose, and mouth were essentially negative. The chest revealed decreased expansion, with a distinct lag on the left side. Auscultation revealed diminished breath sounds over the left upper lobe, with considerable prolongation of expiration. The percussion note was dull over this area and there was no transmitted tactile fremitus. A small mass was palpated in the left supraclavicular fossa, which seemed connected with the deep-lying structures in this portion of the thoracic cage. The abdomen was soft and there were no masses palpable. The liver extended one centimeter below the costal margin. Several examiners believed that the lower pole of the right kidney was palpable.

Fig. 1. The large lesion in the upper lobe with its fairly well defined border is well demonstrated. Note the deviation in the line of the trachea to the right. The smaller metastatic nodules are seen at *A*. The elevation of the left dome of the diaphragm was due to involvement of the phrenic nerve.

The laboratory findings were essentially negative. The patient had a moderate hypochromic anemia with a slight leukocytosis. Blood chemistry examinations were within normal limits and the Wassermann was negative. The urine specific gravity varied between 1.010 and 1.025, and at no time during the patient's illness were red blood cells found in the urine. The phenolsulphonphthalein elimination was 40 per cent during the first hour and 20 per cent during the second hour.

On September 14, a roentgen examination of the chest revealed the following: "There is a mass lesion occupying the upper half of the left chest. It is spherical in configuration, sharply outlined, and extends from the mediastinum to the periphery, laterally and posteriorly. A small amount of fluid is present in the left chest. The upper mediastinum is

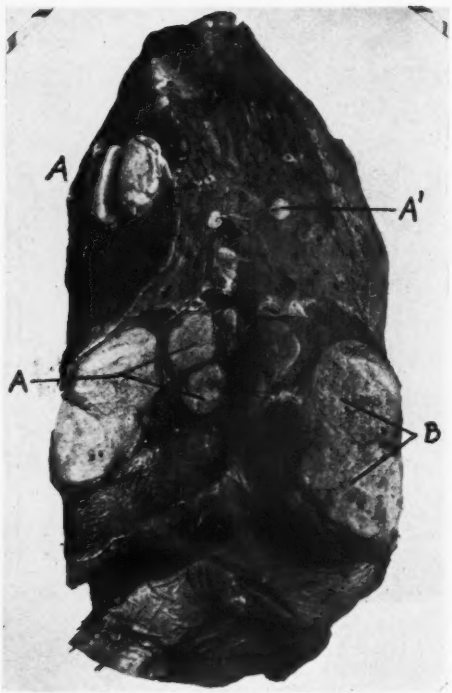


Fig. 2. (Photograph of another case.) *A'* demonstrates the very early type of metastatic invasion. The "cannon ball" is seen at *A*. Note the tendency for the lesions to remain single and discrete. *B* represents the result of coalescence of two "cannon balls," forming the type of lesion found at autopsy in the case reported.

displaced to the right, and the left dome of the diaphragm is elevated and fixed. There are several nodular areas in the right lower lung-field which might be neoplastic or inflammatory in origin." (Fig. 1.)

Dr. Gabriel Tucker bronchoscoped the patient, but was unable to visualize anything abnormal in the bronchial tree with the exception of some fixation of the left side of the lung, which he believed due to a tumor outside of the bronchus.

The clinical diagnostic possibilities included fibroid tuberculosis and primary pulmonary malignancy.

In spite of the predominantly primary pulmonary history and physical findings we could not reconcile ourselves to the idea that the roentgenograms indicated a primary pulmonary process. We believed that the demonstrated lesions were metastatic manifestations of a primary lesion in some other portion of the body. We had no idea as to the nature of the primary malignancy, but believed that it was not primary in the left upper lung-field. While a primary lung tumor with metastases into the contralateral chest was a distinct possibility, we felt that the displacement of the trachea away from the site of maximum involvement mitigated against the diagnosis, since our experience has been that lung tumors producing atelectasis pull the trachea toward the site of involvement unless a fluid collection prevents such tracheal deviation. The third possibility was a primary mediastinal neoplasm, but, in view of the fact that the clinical findings did not suggest this type of lesion, we were inclined to rule it out. An aortic aneurysm was mentioned only to be excluded because of the involved areas in the right lower lung-field. The final radiographic diagnosis was "metastatic malignancy of unknown origin."

Shortly after the roentgenograms were first examined, Dr. Paul Schraer, a member of our staff, suggested that the lung changes were due to metastatic hypernephroma, basing his diagnosis upon the roentgen finding in another patient in whom the diagnosis was subsequently confirmed. Intravenous urography was resorted to in order to aid in establishing a diagnosis, and a definite deformity suggesting a renal tumor involving the right kidney was found. It seemed then that the patient had a primary renal neoplasm, with metastases to both lung-fields.

On September 29, the patient was transfused because his condition had grown increasingly grave. About one hour after the transfusion was completed the patient suddenly became cyanotic and unconscious. In spite of supportive therapy, the patient did not recover and died several days later.

At postmortem examination, the entire upper lobe of the left lung was adherent to the parietal pleura. The left lung weighed 1,320 grams. The lung tissue was edematous, and the lower lobe contained several pea-sized metastatic nodules. The upper lobe was firm and solid. Its cut surface showed a well demarcated mass the size of an orange composed of solid tissue with a necrotic central area. The hilum glands were enlarged and infiltrated. The right lung weighed 820 grams, and it also contained many firm nodules which varied in size from an oat grain to a large lima bean. The entire upper pole of the right kidney was replaced by a large gray mass which merged with another mass the size of a goose egg in the adrenal region. The left kidney was also involved to a lesser degree. There was a large thrombus in the heart from which microscopic sections were obtained and which showed occasional clumps of neoplastic cells. The pathologic diagnosis was: "hypernephroma involv-

ing both kidney regions, with metastases to both lungs."

COMMENT

Usually hypernephromas produce large rounded masses in the lungs, which roentgenographically, are extraordinarily clear-cut in outline. Because of their discrete and spherical appearance, the lesions have frequently been called "cannon ball metastases." The typical "cannon ball" is probably due to a single metastatic nodule, whereas the larger and less clearly defined lesion, demonstrated in our case, represents a group of nodules that have coalesced and formed one large ill-defined mass (Fig. 2).

SUMMARY

The unusual pulmonary manifestations in hypernephroma described in this report have been previously described by Krumbhaar and others. We merely wish to add another case to the few reported with primary pulmonary symptoms in order to stress the bizarre pulmonary picture that may occur in hypernephroma.

MULTIPLE MYELOMA[1]

By V. W. KOCH, M.D., *and* F. H. KUEGLE, M.D.,
Janesville, Wisconsin

Case 1. E. S., aged 49 years, white, a printer by occupation, consulted one of us (V. W. K.) on Feb. 1, 1935. He complained of weakness, shortness of breath on exertion, and pain involving the lower dorsal and upper lumbar segments of the back. This symptom-complex was ushered in in May, 1934, when he experienced a sudden pain in the back in the region of the floating ribs. Since then his symptoms have gradually increased in intensity till now he has difficulty in moving about freely and in getting out of bed. He has had a cough for the past six months but there is no expectoration. Coughing, sneezing, and breathing deeply aggravate his pain. He has lost no weight yet is unable to work because of weakness. He has a whitish precipitate in the urine which is more noticeable toward the end of micturition.

Clinical examination reveals a very pale, well developed male, six feet in height, weighing 180 pounds. His pulse rate is 99; temperature 96; systolic blood pressure 132, diastolic 98. The blood count reveals 10,500 white blood cells, 4,180,000 red blood cells. The hemoglobin is 58 per cent. The differential blood count is as follows: small lymphocytes, 22 per cent; large lymphocytes, 2 per cent; polymorphonuclears, 75 per cent; large mononuclears, 1 per cent.

Chemical examination of the blood reveals

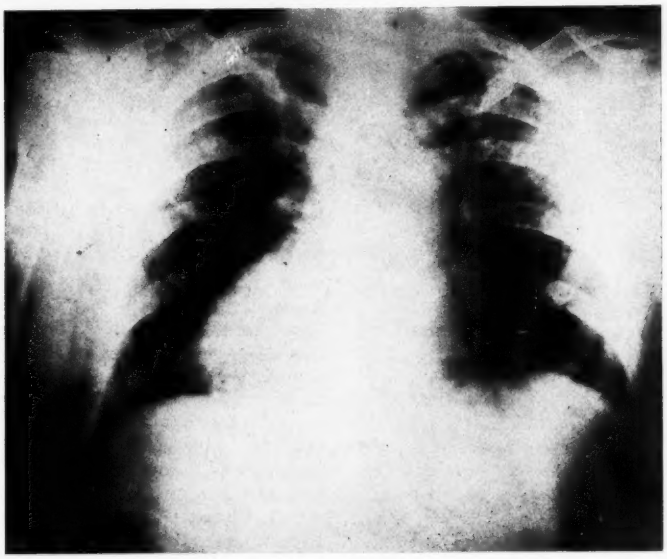


Fig. 1. Reveals osteoporosis of the ribs and the sternal ends of both clavicles.

[1] Read at the Semi-annual Meeting of the Radiological Section of the Wisconsin State Medical Society, Milwaukee, Wisconsin, May 17, 1935.

166 mg. of cholesterol per 100 c.c.; 68 mg. sugar per 100 c c.; 15 mg. calcium per 100 c.c., which is an increase from the average normal

Fig. 2. Reveals marked decalcification of the upper thirds of both radii.

amount of 9 to 12; phosphorus 3 mg. per 100 c.c., which is a relatively low normal amount. The Wassermann reaction is negative.

Gastric analysis reveals the following:

Fasting—0 free HCl—18 c c., total acidity 10
30 minutes—0 free HCl—112 c.c., total acidity 6
1 hour—0 free HCl—20 c.c., total acidity 5

The phenolsulphonphthalein intravenous kidney function test reveals 14 per cent at the 15-minute interval, 13 per cent at 30 minutes, and 15 per cent at one hour.

The water test is shown below.

There is 0.7 per cent urea present at the first hour and 0.9 per cent at the second hour, whereas there normally should be over 2 per cent of the 15 grams given. The urine is cloudy, acid in reaction, and contains 10 per cent albumin. The albumin is of the Bence-Jones variety. In sharp contradistinction to the urine, the blood contains only a small percentage of Bence-Jones albumin.

Fig. 3. Reveals multiple, large and small, round, punched-out areas of rarefaction of the cranium.

The x-ray examination reveals multiple punched-out areas of rarefaction involving the ribs and sternum. The upper thirds of the radii are markedly osteoporotic. The lower ends of the femora contain tiny rarefied areas. The right fibula shows several demineralized areas extending from the medullary canal outward into the cortex. The skull is peppered with punched-out atrophic areas varying in size from a bird shot to a silver dollar.

The x-ray and clinical findings in this case, in which Bence-Jones albuminuria is present in a man 49 years of age, showing marked secondary anemia, suggest the probability that the multiple bone changes described are due to a malignant process but leave one in doubt as to the etiology of the disease. A number of diseases in which multiple, punched-out rarefactions occur in the bones must be considered in the differential diagnosis. These include the following: metastatic carcinomas; multiple sarcomas; osteomalacia; poly-fibrocystic disease; xanthomatosis, and multiple myelomas.

In an exhaustive article on multiple myeloma in the April, 1928, issue of the "Archives of

	8 A.M.	8:30 A.M.	9 A.M.	9:30 A.M.	10 A.M.	10:30 A.M.	11 A.M.	11:30 A.M.	12 NOON
Spec. grav.	1,019	1,025	1,008	1,006	1,005	1,005	1,005	1,010	1,016
Volume	175 c.c.	11 c.c.	115 c.c.	124 c.c.	140 c.c.	127 c.c.	110 c.c.	56 c.c.	36 c.c.
Albumin	+ + +	Q. N. S.	Neg.	Trace	Ft. Trace	Trace	Ft. Trace	H. Trace	+

Surgery," Geschickter and Copeland state that the incidence of bone involvement according to location is as follows in order of frequency:

Fig. 4. Low-power photomicrograph of biopsy tissue removed from gladiolus of sternum.

Fig. 5. High-power photomicrograph of decalcified bone removed from sternum.

lumbar spine; ribs; vault of skull; upper halves of the femora; the upper two-thirds of the humeri; the clavicles; the pelvis, and the lower halves of the femora. In this case the order of frequency is atypical and no changes whatever can be detected in the spine. The bone changes are most pronounced in the upper ends of the radii and in the skull. As regards the significance of Bence-Jones albuminuria, Geschickter and Copeland state that these albuminous bodies in the urine depend on the appearance of a white cloudy precipitate between the temperatures of 50 and 60 degrees C.

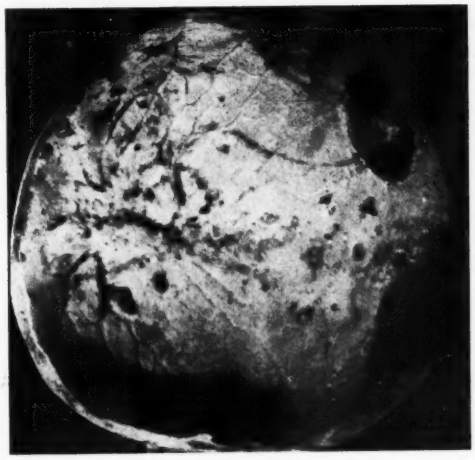


Fig. 6. Photograph of calvarium showing marked bone destruction by myelomas (patient died June 11, 1935).

(sometimes as low as 43 and 46), and on further heating, this precipitate goes into solution at about boiling (90 and 100 degrees C.). On cooling, the precipitate reappears. They estimate the Bence-Jones test to be present in 65 per cent of the cases of myeloma. It may be present in a wide variety of conditions in-

cluding metastatic tumors of bones, multiple sarcoma of bones, senile osteomalacia, von Recklinghausen's disease, comminuted fracture, caries of the spine, tumor of the jaw, lymphatic leukemia, myelogenous leukemia, chloroma, polycythemia, and experimental aplastic anemia (in a dog).

The history of this case, as well as the clinicopathologic and x-ray findings, quite definitely rules out all of these conditions as possibilities to be considered in a diagnosis excepting multiple sarcoma of bones, poly-fibro-cystic disease, and myeloma. The two former diseases occur most commonly before the age of 40, while myeloma is almost always encountered after the age of 40. However, no positive evidence has so far been adduced to label this an undoubted case of myeloma: hence a diagnosis is entirely dependent upon a biopsy.

On March 5, one of us (V. W. K.) removed a button of bone from the sternum. Microscopic examination of this tissue by Dr. W. D. Stovall, pathologist of the University of Wisconsin, establishes a diagnosis of "plasma-cell myeloma of the small cell type." This case is interesting chiefly because of the rareness of the incidence of myeloma. From 1848, when Bence-Jones reported a case of mollities ossium (most likely myeloma) in which a peculiar type of albumin reaction was discovered, which has since then gone under the nomenclature of Bence-Jones albumose, up to 1928, only about 425 cases of myeloma had been reported in medical literature. In a brief article in the March 13, 1926, issue of the "British Medical Journal," Ernest S. Reynolds expresses the opinion that myeloma is more common than is usually supposed, and infers that the disease frequently remains unrecognized.

The prognosis in multiple myeloma is hopeless as no treatment has ever arrested the progress of the disease permanently. Cobra venom administered intravenously is said to relieve pain. Liver therapy may be used advantageously to combat anemia. X-ray irradiations sometimes inhibit the progress of the disease for a considerable length of time. Death may occur at any time within a period of about four years from the onset of the symptoms.

A MULTIPLE X-RAY THERAPY CONE FOR ORIFICIAL AND SMALL FIELD WORK

By JOHN R. CARTY, M.D., *and* E. M. CLAIBORNE, M.D., *New York City*

The shock-proof therapy machines now available have greatly increased the possibilities of external radiation largely because of the increased flexibility which permits of more accurate direction of the radiation. This flexibility also permits one to apply radiation directly into the body cavities, and the use of smaller cones is especially valuable in head and neck work.

With these thoughts in mind, detachable cones and special orificial applicators were designed for external and orificial radiation, using a single cone as a base. The end of a square cone which fitted our outfit was sawed off at a point 40 cm. from the target of the x-ray tube and two grooves were made into which could be slipped the detachable cone. The length of the

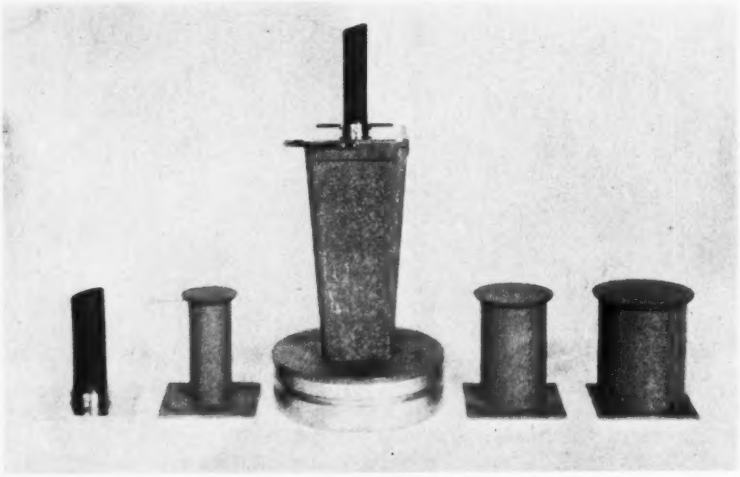


Fig. 1. The permanent base portion of the cone is seen, with an intra-oral applicator partially inserted into the groove. The other detachable cones are shown

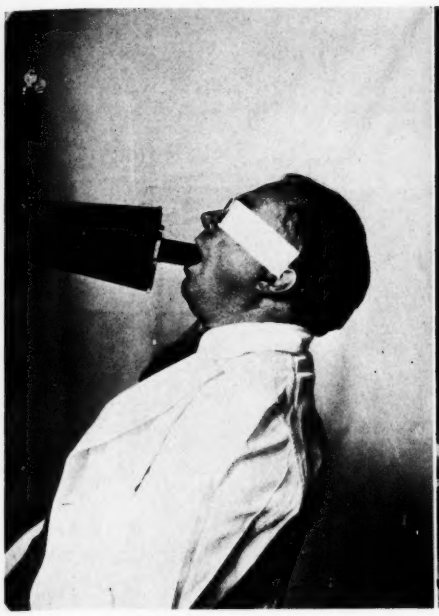


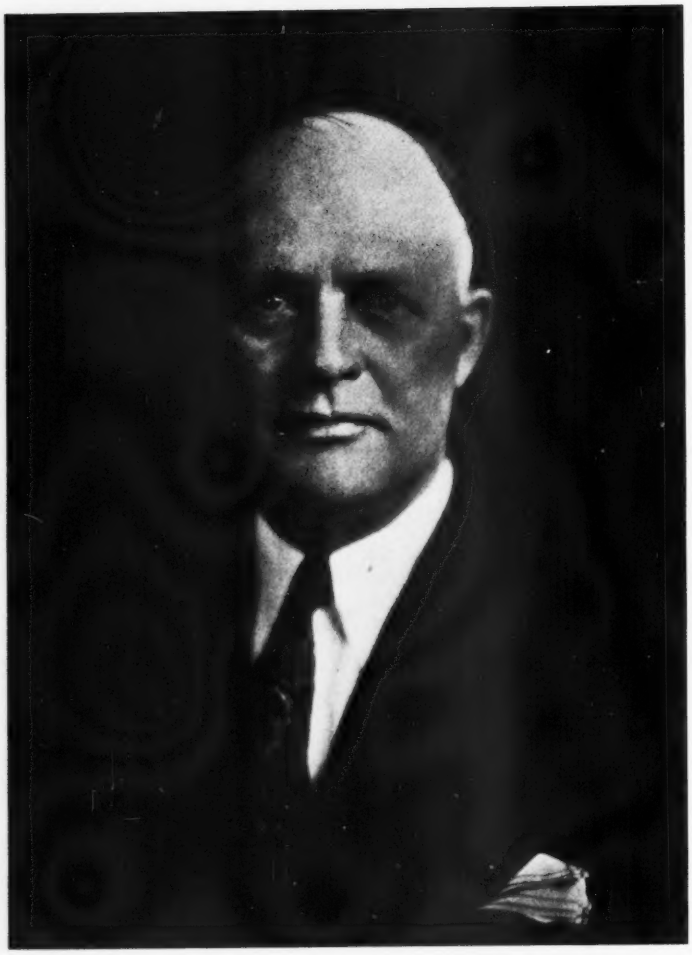
Fig. 2. Showing the application of radiation direct to the tonsillar region. The bakelite tube is seen in the mouth. The head has been turned slightly to one side and the bevelled edge is directly against the right tonsil.

Fig. 3. One of the small cones is in place for radiating the base of the tongue. Note the spring clip which holds the detachable cone in place.

latter was made 10 cm. in order that the total target-skin distance, when in use, would be 50 centimeters. Cones for 80 cm. distance could just as readily be made. The cones for outside application were made of lead with a circular fiber disc at the end, thus permitting pressure to be exerted against the skin without discomfort to the patient. The bases of the detachable cones were made of lead. The intra-oral applicator consisted of a bakelite tube bevelled at one end and inserted at the other into the lead base. The center of the bakelite tube and the center of the base cone were made to coincide exactly. By keeping the bakelite tube small, not to exceed 7 sq. cm., practically speaking, only central radiation is used, the oblique rays being stopped by the lead base. Measurements have shown that there is an escape of from 2 to 3 r per minute from the side of the bakelite tubes, measured in air, which of course is negligible. The tube is bevelled at the end to permit of greater flexibility and more comfort to the patient, particularly as most intra-oral malignancies are apt to lie on either side of the midline.

The intra-oral applicators illustrated here are of 2.3 sq. cm. and 7 sq. cm., while the cones for outside use are 12.5, 28, and 50 sq. cm., respectively, in area. While the total length of the permanent and temporary cone can be made any desired measure, it is best not to shorten the permanent cone too much because there would then be a greater percentage of oblique radiation escaping from the sides of the bakelite tube. The same principle can be applied to intravaginal work for direct application of radiation to the cervix. At present we are working on the design of such a cone.

With this arrangement, a series of small cones of various sizes can be obtained at a relatively small cost and orificial radiation can be safely and conveniently given.



Thomas A. Burcham, M.D., of Des Moines, Iowa, President of the Radiological Society of North America.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

THE ANNUAL MEETING

The Twenty-first Annual Meeting of the Radiological Society of North America, held in Detroit, Michigan, December 2–6, 1935, was one of the Society's most outstanding conventions. The Hotel Statler in Detroit gave superb arrangements and facilities for the physical portion of the convention, allowing the Scientific Exhibits, the Commercial Exhibits, the Registration Desk, and the Scientific Sessions to be placed on the same floor. This entire space was air-conditioned and furnished unusual comfort to those in attendance. Communication over the Public Address System provided simultaneous dissemination of information over the whole convention area. The personnel of the Hotel Statler, from the manager, Mr. J. Henry Pichler, to the bus boys, are to be commended for their contribution to the success of the meeting, by their earnest and whole-hearted co-operation.

Attending members came from all parts of the United States and Canada, increasing the registration over former years. There were also a large number of non-member visitors, many of whom came from great distances.

One complete wing of the convention floor was devoted to Scientific Exhibits, providing material for many hours of valuable study. The prizes for Scientific Awards went to: H. K. Pancoast, M.D., and Associates, University of Pennsylvania, and to the Mayo Foundation for Medical Research, Mayo Clinic. Honorable mention was given Edwin Price Fowler, Jr., M.D., Department of Otolaryngology, Columbia University.

The Commercial Exhibits showed numerous advances in type and style of apparatus and permitted those attending the meeting to have close contact with the manufacturers and observe demonstrations by the factory representatives.

The Scientific Sessions ran five full days of the meeting and the wealth of material presented will be published during the coming year in RADIOLOGY. Professor Hermann Holthusen, M.D., of Hamburg, Germany, a guest speaker at the meeting, presented "The Time Factor in Radiotherapy" as well as "Recent Developments in Radiotherapy." Those attending the meeting also had the privilege of hearing M. Demerec, Ph.D., Carnegie Institute, Cold Spring Harbor, New York, present "Hereditary Effects of Radiation." The scientific papers were extremely well balanced and properly proportioned between therapy and diagnosis.

Because of the need felt for adequate time to study and quiet surroundings in which to concentrate, the clinics were placed in another part of the hotel, at a distance from the noise and confusion of the convention proper. Consequently they were better attended. The material presented was well worth while and because of the change in set-up, members came away with a keener appreciation of what the clinic exhibit has to offer.

The Detroit Roentgen Society and the Wayne County Medical Society contributed no small part to the success of the convention in their excellent social arrangements, as well as the pre-convention and convention preparations.

It is regrettable that illness prevented our President, Lloyd Bryan, M.D., of San Francisco, California, from being present at the convention. His program showed very careful and thoughtful consideration and he is to be congratulated for its excellence and diversification.

DONALD S. CHILDS, M.D.

ANNOUNCEMENTS

THE AMERICAN COLLEGE OF PHYSICIANS

The Twentieth Annual Session of the American College of Physicians will be held in Detroit with headquarters at the Book-Cadillac Hotel, March 2–6, 1936.

Dr. James Alex. Miller, of New York City, is President of the College, and has arranged a program of general scientific sessions of great interest to those engaged in the practice of Internal Medicine and associated specialties. Dr. Charles G. Jennings, of Detroit, is the General Chairman of the Session, and is in charge of the program of clinics and demonstrations in the hospitals, medical schools, and other Detroit institutions. Dr. James D. Bruce, Vice-

president in Charge of University Relations, University of Michigan, is Vice-chairman of the Committee on Arrangements, and has in charge the preparation of an all-day program to be conducted at the University of Michigan on Wednesday, March 4. Dr. Walter B. Cannon, Professor of Physiology at Harvard University Medical School, will deliver the annual Convocation Oration on "The Rôle of Emotion in Disease." Dr. Miller's presidential address will be on "The Changing Order in Medicine." About fifty eminent authorities will present papers at the general scientific sessions, while clinics and demonstrations will be conducted at the Harper, Receiving, Ford, Grace, Herman Kiefer, and Children's Hospitals of Detroit.

DIPLOMATES OF THE AMERICAN BOARD OF RADIOLOGY

(January 1, 1936)

The following is a complete list of the radiologists who have appeared before the Board and passed a satisfactory examination in the field indicated.

	Name	Address	Field
1.	Alexander, Fay K.	Philadelphia, Pa.	Radiology
2.	Allen, B. M.	Wilmington, Del.	Diagnostic Roentgenology
*3.	Allen, Bundy	Tampa, Fla.	Radiology
4.	Allen, Kenneth D. A.	Denver, Col.	Radiology
5.	Allen, Lewis G.	Kansas City, Kan.	Radiology
6.	Allen, M. Lowry	Philadelphia, Pa.	Radiology
7.	Allen, William E., Jr.	St. Louis, Mo.	Roentgenology
8.	Alley, Reuben G.	Pittsburgh, Pa.	Diagnostic Roentgenology
9.	Allison, R. G.	Minneapolis, Minn.	Roentgenology
10.	Altman, William S.	Quincy, Mass.	Radiology
11.	Ané, Joseph N.	New Orleans, La.	Radiology
12.	Anspach, William E.	Chicago, Ill.	Roentgenology
13.	Archer, Vincent W.	University, Va.	Roentgenology
14.	Arens, Robert A.	Chicago, Ill.	Radiology
15.	Ashbury, Howard E.	Baltimore, Md.	Roentgenology
16.	Atkins, Samuel M.	Waterbury, Conn.	Roentgenology
17.	Aurelius, J. Richards, Jr.	St. Paul, Minn.	Roentgenology
18.	Avery, Philip S.	New Brunswick, N. J.	Roentgenology
19.	Bachman, M. H.	Youngstown, O.	Roentgenology
20.	Bader, Ellis R.	Cincinnati, O.	Radiology
21.	Bailey, Wilbur	Pasadena, Cal.	Radiology
22.	Baker, Charles F.	Newark, N. J.	Roentgenology
23.	Baker, Edgar C.	Youngstown, O.	Radiology
24.	Ball, Robert P.	Chattanooga, Tenn.	Diagnostic Roentgenology
25.	Barfield-Carter, M.	Birmingham, Ala.	Roentgenology
26.	Barker, W. Allen	Petersburg, Va.	Roentgenology
27.	Barnes, John M.	Buffalo, N. Y.	Roentgenology
28.	Barnett, Arthur F.	Eugene, Ore.	Radiology
29.	Barrow, S. C.	Shreveport, La.	Radiology
30.	Barth, Earl E.	Chicago, Ill.	Roentgenology
31.	Bauer, August A.	Chicago, Ill.	Roentgenology
32.	Baum, Samuel M.	New York, N. Y.	Therapeutic Radiology
33.	Baxter, O. D.	Sumter, S. C.	Radiology
34.	Beeler, Raymond C.	Indianapolis, Ind.	Radiology
35.	Beilin, David S.	Chicago, Ill.	Radiology
36.	Bell, Joseph C.	Louisville, Ky.	Roentgenology
*37.	Benishek, Werner L.	Aurora, Ill.	Roentgenology
38.	Benjamin, Emanuel W.	Providence, R. I.	Radiology
39.	Berg, H. Milton	Bismarck, N. D.	Radiology
40.	Bernstein, Joshua H.	New York, N. Y.	Radiology
41.	Bethea, William R.	Memphis, Tenn.	Roentgenology
42.	Birkelo, Carl C.	Detroit, Mich.	Roentgenology
43.	Bishop, Paul A.	Philadelphia, Pa.	Radiology
44.	Blackett, Charles W.	Boston, Mass.	Roentgenology
45.	Blaine, Edward S.	Los Angeles, Cal.	Roentgenology
46.	Bliss, Gerald D.	Altoona, Pa.	Radiology
47.	Bogan, Isabel K.	Boston, Mass.	Roentgenology
48.	Bogan, Mary E.	Brookline, Mass.	Roentgenology
49.	Bogart, Franklin B.	Chattanooga, Tenn.	Roentgenology
50.	Bonnar, James M.	New Bedford, Mass.	Radiology
51.	Bonoff, Karl M.	Los Angeles, Cal.	Diagnostic Roentgenology
52.	Borzell, Francis F.	Philadelphia, Pa.	Radiology

*Deceased.

53.	Bowen, Carl B.	Oakland, Cal.	Roentgenology
54.	Bowen, David R.	Philadelphia, Pa.	Radiology
55.	Bowing, Harry H.	Rochester, Minn.	Therapeutic Radiology
56.	Boyd, James F.	Providence, R. I.	Radiology
57.	Boyes, James G.	Plainfield, N. J.	Roentgenology
58.	Bradley, Robert A.	Atlantic City, N. J.	Radiology
59.	Brams, Julius	Chicago, Ill.	Radiology
60.	Brenneman, Richard E.	Meadville, Pa.	Radiology
61.	Bridenbaugh, J. H.	Billings, Mont.	Radiology
62.	Broeser, Henry V.	Hoboken, N. J.	Diagnostic Roentgenology
63.	Bromer, Ralph S.	Bryn Mawr, Pa.	Radiology
64.	Brooksher, W. R.	Fort Smith, Ark.	Radiology
65.	Brouse, Ivan E.	Jacksonville, Ill.	Roentgenology
66.	Brown, H. O.	Tampa, Fla.	Roentgenology
67.	Brown, Percy	Boston, Mass.	Roentgenology
68.	Brown, Samuel	Cincinnati, O.	Roentgenology
69.	Bruck, Samuel	Philadelphia, Pa.	Radiology
70.	Bryan, Lloyd	San Francisco, Cal.	Roentgenology
71.	Burcham, Thomas A.	Des Moines, Ia.	Radiology
72.	Burnett, Harry W.	Dayton, O.	Roentgenology
73.	Butler, Patrick F.	Boston, Mass.	Radiology
74.	Caldwell, Charles S.	Pittsburgh, Pa.	Roentgenology
75.	Camp, John D.	Rochester, Minn.	Roentgenology
76.	Capp, Charles S.	San Antonio, Texas	Radiology
77.	Carlson, Glenn D.	Dallas, Texas	Roentgenology
78.	Carr, Edson W.	Chicago, Ill.	Diagnostic Roentgenology
79.	Carter, Ray A.	Los Angeles, Cal.	Roentgenology
80.	Carty, John R.	New York, N. Y.	Radiology
81.	Case, James T.	Chicago, Ill.	Radiology
82.	Cathcart, John W.	El Paso, Texas	Radiology
83.	Challenger, Chester J.	Chicago, Ill.	Roentgenology
84.	Chamberlain, W. Edward	Philadelphia, Pa.	Radiology
85.	Chapman, John F.	Pasadena, Cal.	Diagnostic Roentgenology
86.	Childe, Arthur E.	Montreal, Que.	Diagnostic Roentgenology
87.	Childs, Donald S.	Syracuse, N. Y.	Roentgenology
88.	Christie, Arthur C.	Washington, D. C.	Radiology
89.	Cilley, Earl I. L.	Bellingham, Wash.	Radiology
90.	Claiborne, Elmer M.	New York, N. Y.	Radiology
91.	Clark, Daniel M.	Santa Barbara, Cal.	Radiology
92.	Clark, James J.	Atlanta, Ga.	Roentgenology
93.	Clark, Stanley A.	South Bend, Ind.	Radiology
94.	Cleaves, Edwin N.	Boston, Mass.	Diagnostic Roentgenology
95.	Cleghorn, Charles D.	Miami, Fla.	Radiology
96.	Clement, Gage	Duluth, Minn.	Radiology
97.	Cleveland, Walter R.	Evansville, Ind.	Radiology
98.	Coate, Joseph D.	Oakland, Cal.	Radiology
99.	Coe, Frederick O.	Washington, D. C.	Radiology
100.	Coffin, Whitman K.	Boston, Mass.	Roentgenology
101.	Cohoon, Carl W.	Bay Shore, N. Y.	Diagnostic Roentgenology
102.	Cole, John M.	Windsor, Ont.	Roentgenology
103.	Cole, Lewis Gregory	New York, N. Y.	Roentgenology
104.	Cole, Paul F.	Springfield, Mo.	Radiology
105.	Cole, William Gregory	New York, N. Y.	Roentgenology
106.	Coley, Stephen W.	Memphis, Tenn.	Roentgenology
107.	Collins, E. N.	Cleveland, O.	Diagnostic Roentgenology
108.	Collins, James N.	Indianapolis, Ind.	Radiology
109.	Collins, John J.	Thomasville, Ga.	Radiology
110.	Cook, Philip H.	Worcester, Mass.	Radiology
111.	Cooley, Randall M.	Jackson, Mich.	Diagnostic Roentgenology
112.	Coray, Q. B.	Salt Lake City, Utah	Roentgenology
113.	Corcoran, William J.	Scranton, Pa.	Roentgenology
114.	Costolow, William E.	Los Angeles, Cal.	Therapeutic Radiology
115.	Crain, Carroll F.	Corpus Christi, Texas	Radiology
116.	Cramp, George W.	Brooklyn, N. Y.	Roentgenology
117.	Crane, Augustus W.	Kalamazoo, Mich.	Roentgenology
118.	Crossan, John W.	Los Angeles, Cal.	Diagnostic Roentgenology
119.	Crowder, E. R.	Chicago, Ill.	Roentgenology
120.	Cunningham, Lester W.	Mandarin, Fla.	Roentgenology
121.	Currin, Francis W.	Brooklyn, N. Y.	Roentgenology
122.	Cushway, B. C.	Chicago, Ill.	Radiology
123.	Dann, David S.	Kansas City, Mo.	Radiology
124.	Danzer, Joseph T.	Oil City, Pa.	Radiology
125.	Davidson, Sol C.	Rochester, N. Y.	Roentgenology
126.	Davis, Ernest L.	Springfield, Mass.	Roentgenology
127.	Davis, Harold E.	Chicago, Ill.	Radiology
128.	Davis, Henry B.	Lancaster, Pa.	Radiology
129.	Davis, Kenneth S.	Los Angeles, Cal.	Roentgenology

130.	Davison, Chester O.	Poughkeepsie, N. Y.	Radiology
131.	Davison, R. Winthrop	Trenton, N. J.	Roentgenology
132.	Debbie, Anthony G.	St. Albans, N. Y.	Diagnostic Roentgenology
133.	Decker, Fred H.	Peoria, Ill.	Radiology
134.	Dempster, James H.	Detroit, Mich.	Diagnostic Roentgenology
135.	Derr, John S.	Frederick, Md.	Roentgenology
136.	Desjardins, A. U.	Rochester, Minn.	Radiology
137.	Deweese, Everett R.	Kansas City, Mo.	Radiology
138.	Dick, Paul G.	Chicago, Ill.	Roentgenology
139.	Dickinson, J. C.	Tampa, Fla.	Roentgenology
140.	Dixon, George S.	New York, N. Y.	Diagnostic Roentgenology
141.	Donaldson, S. W.	Ann Arbor, Mich.	Roentgenology
142.	Doub, Howard P.	Detroit, Mich.	Radiology
143.	Doughty, William M.	Cincinnati, O.	Radiology
144.	Downing, Robert E.	Terre Haute, Ind.	Diagnostic Roentgenology
145.	Downs, Elwood E.	Woodbury, N. J.	Radiology
146.	Drane, Robert	Savannah, Ga.	Roentgenology
147.	Dresser, Richard	Boston, Mass.	Radiology
148.	Duckworth, Roy D.	White Plains, N. Y.	Roentgenology
149.	Duckworth, Willard D.	New Rochelle, N. Y.	Roentgenology
150.	Dunham, H. Kennon	Cincinnati, O.	Diagnostic Roentgenology
151.	Durrance, Fred Y.	Houston, Texas	Diagnostic Roentgenology
152.	Eastmond, Charles	Brooklyn, N. Y.	Roentgenology
153.	Edeiken, Louis	Philadelphia, Pa.	Radiology
154.	Edwards, Harold G. F.	Shreveport, La.	Radiology
155.	Edwards, J. Bennett	Englewood, N. J.	Roentgenology
156.	Ehrenpreis, Bernard	Brooklyn, N. Y.	Roentgenology
157.	Ehrlich, David E.	New York, N. Y.	Diagnostic Roentgenology
158.	Eldridge, Watson W., Jr.	Washington, D. C.	Roentgenology
159.	Eley, Clayton W.	Norfolk, Va.	Roentgenology
160.	Ellis, Ivan G.	Madison, Wis.	Roentgenology
161.	Elward, Joseph F.	Washington, D. C.	Roentgenology
162.	Eneboe, John B.	San Diego, Cal.	Radiology
163.	Ericksen, Lester G.	Dubuque, Ia.	Radiology
164.	Ernst, Edwin C.	St. Louis, Mo.	Radiology
165.	Erskine, Arthur W.	Cedar Rapids, Ia.	Radiology
166.	Evans, William A.	Detroit, Mich.	Radiology
167.	Eveleth, Fred S.	Concord, N. H.	Roentgenology
168.	Exner, Frederick B.	Bellingham, Wash.	Radiology
169.	Farmer, Harry L.	Cleveland, O.	Radiology
170.	Farrell, John T., Jr.	Philadelphia, Pa.	Roentgenology
171.	Feaster, O. O.	St. Petersburg, Fla.	Radiology
172.	Ferguson, James W.	Pittsburgh, Pa.	Radiology
173.	Fineman, Solomon	New York, N. Y.	Roentgenology
174.	Firor, Whitmer B.	Baltimore, Md.	Roentgenology
175.	Fisher, J. W.	Pittsburgh, Pa.	Radiology
176.	Fisher, Lawrence F.	South Bend, Ind.	Roentgenology
177.	Flinn, F.	Decatur, Ill.	Radiology
178.	Flynn, James M.	Rochester, N. Y.	Radiology
179.	Foley, Joseph M.	Chicago, Ill.	Roentgenology
180.	Ford, Frances A.	Detroit, Mich.	Radiology
181.	Fray, Walter W.	Rochester, N. Y.	Radiology
182.	Freedman, Eugene	Cleveland, O.	Roentgenology
183.	Freedman, John	Detroit, Mich.	Roentgenology
184.	Freid, Jacob R.	New York, N. Y.	Radiology
185.	Frere, John M.	Chattanooga, Tenn.	Roentgenology
186.	Fricke, Robert E.	Rochester, Minn.	Therapeutic Radiology
187.	Friedman, Asa B.	Brooklyn, N. Y.	Radiology
188.	Friedman, Harry F.	Boston, Mass.	Radiology
189.	Friedman, Jacob	New York, N. Y.	Roentgenology
190.	Friedman, Milton	Newark, N. J.	Therapeutic Radiology
191.	Fruchter, Joseph M.	Philadelphia, Pa.	Radiology
192.	Fugate, Isaac T.	Louisville, Ky.	Radiology
193.	Fulmer, Charles C.	San Francisco, Cal.	Roentgenology
194.	Fulton, Huston F.	Columbus, O.	Diagnostic Roentgenology
195.	Furey, Warren W.	Chicago, Ill.	Radiology
196.	Furst, Nathan J.	Newark, N. J.	Diagnostic Roentgenology
197.	Garland, L. H.	San Francisco, Cal.	Radiology
198.	Gates, Russell	Santa Barbara, Cal.	Roentgenology
199.	Gemmell, J. H.	Philipsburg, Pa.	Roentgenology
200.	George, Arial W.	Boston, Mass.	Roentgenology
201.	Gerber, Isaac	Providence, R. I.	Radiology
202.	Gershon-Cohen, Jacob	Philadelphia, Pa.	Radiology
203.	Geyman, Milton J.	Santa Barbara, Cal.	Diagnostic Roentgenology
204.	Ghrist, David M.	Glendale, Cal.	Roentgenology
205.	Gianturco, Cesare	Urbana, Ill.	Roentgenology

206.	Giles, Roy G.	Temple, Texas	Roentgenology
207.	Gillies, Carl L.	Iowa City, Ia.	Radiology
208.	Gilmore, William M.	Stratford, Ont.	Roentgenology
209.	Gingold, Joseph R.	Meadville, Pa.	Roentgenology
210.	Glover, Milton H.	Wichita Falls, Texas	Radiology
211.	Goin, Lowell S.	Los Angeles, Cal.	Roentgenology
212.	Golden, Ross	New York, N. Y.	Roentgenology
213.	Goldsmith, Maurice F.	Pittsburgh, Pa.	Roentgenology
214.	Goodrich, Murray E.	Toledo, O.	Radiology
215.	Goodwin, Perry B.	Peoria, Ill.	Radiology
216.	Gorsline, Clarence S.	Battle Creek, Mich.	Roentgenology
217.	Granger, Amédée	New Orleans, La.	Diagnostic Roentgenology
218.	Gray, Horace D.	Memphis, Tenn.	Radiology
219.	Grier, George W.	Pittsburgh, Pa.	Radiology
220.	Groeschel, L. B.	New York, N. Y.	Radiology
221.	Groover, Thomas A.	Washington, D. C.	Radiology
222.	Habbe, J. Edwin	Milwaukee, Wis.	Roentgenology
223.	Hall, E. Walter	Detroit, Mich.	Radiology
224.	Hall, Wendell C.	Philadelphia, Pa.	Radiology
225.	Hankins, John L.	Johnson City, Tenn.	Roentgenology
226.	Hanson, Cyrus O.	Minot, N. D.	Radiology
227.	Harding, Donnan B.	Lexington, Ky.	Radiology
228.	Hare, Hugh F.	Boston, Mass.	Radiology
229.	Harrington, B. D.	Tacoma, Wash.	Radiology
230.	Harris, Clarence P.	Houston, Texas	Roentgenology
231.	Harris, John H.	Harrisburg, Pa.	Radiology
232.	Harris, Milo T.	Spokane, Wash.	Radiology
233.	Harris, William	New York, N. Y.	Therapeutic Radiology
234.	Hartung, Adolph	Chicago, Ill.	Roentgenology
235.	Hasley, Clyde K.	Detroit, Mich.	Radiology
236.	Hauser, Harry	Cleveland, O.	Radiology
237.	Hawley, Sydney J.	Danville, Pa.	Roentgenology
238.	Haworth, Wallace	Portland, Ore.	Radiology
239.	Hay, Percy D., Jr.	Florence, S. C.	Radiology
240.	Heacock, Charles H.	Memphis, Tenn.	Roentgenology
241.	Healy, Thomas R.	Boston, Mass.	Roentgenology
242.	Heatley, John E.	Oklahoma City, Okla.	Diagnostic Roentgenology
243.	Heberding, John	Youngstown, O.	Radiology
244.	Hefke, Hans W.	Milwaukee, Wis.	Radiology
245.	Hendricks, Elliott M.	Fort Lauderdale, Fla.	Roentgenology
246.	Hendrickson, Anna R.	Canton, O.	Roentgenology
247.	Henry, Lucas S.	Syracuse, N. Y.	Roentgenology
248.	Herpel, Frederick K.	West Palm Beach, Fla.	Roentgenology
249.	Herrmann, William G.	Asbury Park, N. J.	Radiology
250.	Hess, George H.	Uniontown, Pa.	Roentgenology
251.	Heylmun, Harry H.	Long Beach, Cal.	Diagnostic Roentgenology
252.	Hill, Harold A.	San Francisco, Cal.	Radiology
253.	Hill, Walter C.	Cleveland, O.	Roentgenology
254.	Hilt, Lawrence M.	Springfield, Ill.	Roentgenology
255.	Hilton, James M.	Klamath Falls, Ore.	Roentgenology
256.	Hirsch, I. Seth	New York, N. Y.	Radiology
257.	Hodges, Fred J.	Ann Arbor, Mich.	Radiology
258.	Hodges, Fred M.	Richmond, Va.	Radiology
259.	Hodges, Paul C.	Chicago, Ill.	Radiology
260.	Hoffman, William J.	New York, N. Y.	Therapeutic Radiology
261.	Holly, Leland E.	Muskegon, Mich.	Roentgenology
262.	Holmes, George W.	Boston, Mass.	Radiology
263.	Holmes, Ralph W.	Chillicothe, O.	Roentgenology
264.	Holston, Joel D.	Massillon, O.	Roentgenology
265.	Hopkirk, C. C.	Santa Monica, Cal.	Roentgenology
266.	Horrigan, Arthur J.	Springfield, Mass.	Roentgenology
267.	Howard, William P.	Albany, N. Y.	Roentgenology
268.	Howes, William E.	Brooklyn, N. Y.	Radiology
269.	Hubeny, M. J.	Chicago, Ill.	Roentgenology
270.	Hufford, Clarence E.	Toledo, O.	Radiology
271.	Hunt, Howard B.	Omaha, Neb.	Radiology
272.	Hunt, Russell R.	Providence, R. I.	Roentgenology
273.	Hunter, James W., Jr.	Norfolk, Va.	Radiology
274.	Hutton, Frederick C.	Philadelphia, Pa.	Roentgenology
275.	Hynes, William P.	Washington, D. C.	Therapeutic Radiology
276.	Ikeda, Kano	St. Paul, Minn.	Roentgenology
277.	Illick, H. Earl	New York, N. Y.	Roentgenology
278.	Imboden, Harry M.	New York, N. Y.	Roentgenology
279.	Ingber, Irving S.	San Francisco, Cal.	Radiology
280.	Ivey, H. B.	Goldsboro, N. C.	Radiology
281.	Jaches, Leopold	New York, N. Y.	Radiology

282.	Jackson, Byron H.	Scranton, Pa.	Radiology
283.	Jackson, John B.	Kalamazoo, Mich.	Roentgenology
284.	Jacobs, Lewis G., Jr.	San Francisco, Cal.	Radiology
285.	Jacox, Harold W.	Ann Arbor, Mich.	Radiology
286.	Jarre, Hans A.	Detroit, Mich.	Radiology
287.	Jenkinson, David L.	Chicago, Ill.	Roentgenology
288.	Jenkinson, E. L.	Chicago, Ill.	Radiology
289.	Johannesson, Carl J.	Walla Walla, Wash.	Roentgenology
290.	Johnson, Clayton R.	Los Angeles, Cal.	Roentgenology
291.	Johnson, Ellsworth	Winchester, Va.	Roentgenology
292.	Johnson, Jesse B.	Galveston, Texas	Radiology
293.	Johnson, Vincent C.	Ann Arbor, Mich.	Radiology
294.	Johnston, Zoe Allison	Pittsburgh, Pa.	Therapeutic Radiology
295.	Jones, Horace C.	Detroit, Mich.	Radiology
296.	Jones, William A.	Kingston, Ont.	Radiology
297.	Judy, William S.	Greenville, S. C.	Radiology
298.	Kabakjian, Armen E.	Lancaster, Pa.	Radiology
299.	Kahn, Max	Baltimore, Md.	Roentgenology
300.	Kalbfleisch, William K.	Wheeling, W. Va.	Roentgenology
301.	Kann, U. S.	Binghamton, N. Y.	Radiology
302.	Kaplan, Ira I.	New York, N. Y.	Therapeutic Radiology
303.	Kaplan, Morris	New York, N. Y.	Diagnostic Roentgenology
304.	Kasabach, Haig H.	New York, N. Y.	Radiology
305.	Keichline, John M.	Huntingdon, Pa.	Radiology
306.	Keith, David Y.	Louisville, Ky.	Radiology
307.	Kelly, James F.	Omaha, Neb.	Radiology
308.	Kenning, John C.	Detroit, Mich.	Radiology
309.	Kerby, James P.	Salt Lake City, Utah	Roentgenology
310.	Kerr, H. Dabney	Iowa City, Ia.	Radiology
311.	Kesmodel, Karl F.	Birmingham, Ala.	Radiology
312.	Kidder, Frank W.	Los Angeles, Cal.	Diagnostic Roentgenology
313.	Kinney, Lyell C.	San Diego, Cal.	Radiology
314.	Kirkland, A. Stanley	St. John, N. B.	Roentgenology
315.	Kirklin, B. R.	Rochester, Minn.	Radiology
316.	Klein, William	New Brunswick, N. J.	Radiology
317.	Koiransky, Gennady H.	Long Island City, N. Y.	Diagnostic Roentgenology
318.	Kolvoord, Theodore	Battle Creek, Mich.	Diagnostic Roentgenology
319.	Kornblum, Karl	Philadelphia, Pa.	Radiology
320.	Kuegle, F. H.	Janesville, Wis.	Roentgenology
321.	Lafferty, R. H.	Charlotte, N. C.	Radiology
322.	Landau, George M.	Chicago, Ill.	Roentgenology
323.	Landes, W. L. S.	York, Pa.	Roentgenology
324.	Langan, Paul C.	Akron, O.	Diagnostic Roentgenology
325.	Lange, Sidney	Cincinnati, O.	Radiology
*326.	Langer, Heinz	Pittsburgh, Pa.	Therapeutic Radiology
327.	Larkey, Charles J.	Bayonne, N. J.	Diagnostic Roentgenology
328.	Lattman, Isidore	Washington, D. C.	Radiology
329.	Law, F. M.	New York, N. Y.	Diagnostic Roentgenology
330.	Lawrence, W. S.	Memphis, Tenn.	Radiology
331.	Lawson, John D.	Sacramento, Cal.	Radiology
332.	Leddy, Eugene T.	Rochester, Minn.	Radiology
333.	Ledoux, Alfred C.	Evanston, Ill.	Radiology
334.	Leef, Edward	San Francisco, Cal.	Radiology
335.	LeFevre, Walter I.	Cleveland, O.	Roentgenology
336.	Leibert, Harry F.	Bethlehem, Pa.	Roentgenology
337.	Lenz, Maurice	New York, N. Y.	Therapeutic Radiology
338.	Leonard, Ralph D.	Boston, Mass.	Roentgenology
339.	Leucutia, T.	Detroit, Mich.	Radiology
340.	Levene, George	Boston, Mass.	Roentgenology
341.	Levi, Irwin P.	Anniston, Ala.	Roentgenology
342.	Le Vine, David	El Dorado, Ark.	Diagnostic Roentgenology
343.	Levine, Samuel	Philadelphia, Pa.	Radiology
344.	Levitin, Joseph	San Francisco, Cal.	Radiology
345.	Levyn, Lester	Buffalo, N. Y.	Diagnostic Roentgenology
346.	Lewis, Raymond W.	New York, N. Y.	Diagnostic Roentgenology
347.	Lhevine, Morris B.	Tulsa, Okla.	Roentgenology
348.	Liebman, Charles	Montreal, Que.	Roentgenology
349.	Liljedahl, Elmer N.	Los Angeles, Cal.	Roentgenology
350.	Lingeman, Leslie R.	Rochester, N. Y.	Roentgenology
351.	Lipschultz, Oscar	Minneapolis, Minn.	Roentgenology
352.	Litschgi, Joseph J.	Chicago, Ill.	Roentgenology
353.	Littig, Lawrence V.	Madison, Wis.	Radiology
354.	Lochry, Ralph L.	Indianapolis, Ind.	Roentgenology
355.	Lockwood, Ira H.	Kansas City, Mo.	Radiology
356.	Loud, Norman W.	New Britain, Conn.	Diagnostic Roentgenology

* Deceased.

357.	Lucinian, Joseph H.	Miami, Fla.	Roentgenology
358.	Lutz, Jeremiah F.	York, Pa.	Roentgenology
359.	MacColl, D. R.	Los Angeles, Cal.	Roentgenology
360.	MacMillan, A. S.	Boston, Mass.	Diagnostic Roentgenology
361.	McCarthy, Justin E.	Cincinnati, O.	Radiology
362.	McCaw, William W.	Washington, D. C.	Radiology
363.	McClanahan, Charles W.	West Los Angeles, Cal.	Radiology
364.	McClelland, Donald C.	Lafayette, Ind.	Radiology
365.	McClure, Christopher C.	Nashville, Tenn.	Radiology
366.	McCormick, Arthur F.	Du Bois, Pa.	Roentgenology
367.	McCormick, H. G.	Laurel, Miss.	Roentgenology
368.	McCormick, William M.	Du Bois, Pa.	Roentgenology
369.	McCullough, John F.	Pittsburgh, Pa.	Radiology
370.	McDeed, Winfield G.	Houston, Texas	Roentgenology
371.	McDermott, Joseph L.	Kansas City, Mo.	Radiology
372.	McElfatrick, George C.	Wilmington, Del.	Roentgenology
373.	McEuen, Harry B.	Jacksonville, Fla.	Radiology
374.	McGee, Harry H.	Savannah, Ga.	Roentgenology
375.	McGehee, William H.	Fresno, Cal.	Roentgenology
376.	McGuffin, W. Herbert	Calgary, Alberta	Radiology
377.	McHenry, Rupert K.	Houston, Texas	Roentgenology
378.	McIntosh, Harriet C.	New York, N. Y.	Roentgenology
379.	McKinney, Joseph T.	Roanoke, Va.	Roentgenology
380.	McNabb, Atholl M.	Ottawa, Ont.	Roentgenology
381.	McNamee, Edgar P.	Cleveland, O.	Diagnostic Roentgenology
382.	McNutt, John R.	Duluth, Minn.	Radiology
383.	McPeak, Clarence N.	Fitchburg, Mass.	Roentgenology
384.	McPeak, Edgar M.	Washington, D. C.	Radiology
385.	Magruder, L. Freeland	Norfolk, Va.	Radiology
386.	Mahrer, Herbert A.	Cleveland, O.	Diagnostic Roentgenology
387.	Maier, Roe J.	Chicago, Ill.	Radiology
388.	Malcolmson, Patrick H.	Edmonton, Alberta	Radiology
389.	Malone, Leander A.	Terre Haute, Ind.	Radiology
390.	Mandeville, Frederick B.	Richmond, Va.	Roentgenology
391.	Manges, Willis F.	Philadelphia, Pa	Radiology
392.	Marchbanks, Stanton S.	Chattanooga, Tenn.	Radiology
393.	Marks, Joseph H.	Fall River, Mass.	Roentgenology
394.	Marquis, W. James	Newark, N. J.	Roentgenology
395.	Martin, Charles L.	Dallas, Texas	Radiology
396.	Martin, James M.	Dallas, Texas	Radiology
397.	Martin, William C.	Louisville, Ky.	Roentgenology
398.	Mason, Claude H.	El Paso, Texas	Diagnostic Roentgenology
399.	Masterson, John J.	Brooklyn, N. Y.	Roentgenology
400.	Mattick, Walter L.	Buffalo, N. Y.	Therapeutic Radiology
401.	Maver, William W.	Jersey City, N. J.	Roentgenology
402.	Mawhinney, Harvey N.	Pittsburgh, Pa.	Diagnostic Roentgenology
403.	May, Ernst A.	East Orange, N. J.	Radiology
404.	May, Raymond V.	Cleveland, O.	Radiology
405.	May, Robert J.	Cleveland, O.	Radiology
406.	Mayfield, Claud	Long Beach, Cal.	Diagnostic Roentgenology
407.	Meadows, James A.	Birmingham, Ala.	Radiology
408.	Medelman, John P.	Lubbock, Texas	Roentgenology
409.	Meland, Orville N.	Los Angeles, Cal.	Therapeutic Radiology
410.	Mendelson, Emanuel	Brooklyn, N. Y.	Roentgenology
411.	Menees, Thomas O.	Grand Rapids, Mich.	Radiology
412.	Menville, Leon J.	New Orleans, La.	Radiology
413.	Merchant, Albert K.	Philadelphia, Pa.	Radiology
414.	Merrill, E. Forrest	New York, N. Y.	Roentgenology
415.	Meyer, Keith T.	Evansville, Ind.	Roentgenology
416.	Meyer, William H.	New York, N. Y.	Radiology
417.	Milkman, Louis A.	Scranton, Pa.	Roentgenology
418.	Millwee, Robert H.	Dallas, Texas	Radiology
419.	Ming, Charles M.	Okmulgee, Okla.	Radiology
420.	Minor, Edward G.	Detroit, Mich.	Diagnostic Roentgenology
421.	Moloney, Albert M.	Boston, Mass.	Roentgenology
422.	Moore, Alexander B.	Washington, D. C.	Radiology
423.	Moore, Claude	Washington, D. C.	Radiology
424.	Moore, Daniel M.	Monroe, La.	Roentgenology
425.	Moore, John J.	San Francisco, Cal.	Radiology
426.	Moore, Paul D.	Muncie, Ind.	Radiology
427.	Moore, Sherwood	St. Louis, Mo.	Radiology
428.	Moore, Vernor M.	Grand Rapids, Mich.	Radiology
429.	Morrison, Murray C.	London, Ont.	Radiology
430.	Morrison, Sidney L.	Boston, Mass.	Roentgenology
431.	Morse, Russell W.	Minneapolis, Minn.	Roentgenology
432.	Morton, S. A.	Milwaukee, Wis.	Radiology

433.	Moxness, Bennie A.	Coatesville, Pa.	Diagnostic Roentgenology
434.	Mulligan, Peter B.	Ashland, Pa.	Roentgenology
435.	Murphy, G. W.	Asheville, N. C.	Roentgenology
436.	Murphy, John T.	Toledo, O.	Radiology
437.	Myers, Ralph E.	Oklahoma City, Okla.	Radiology
438.	Nathanson, Louis	Brooklyn, N. Y.	Radiology
439.	Nessa, N. J.	Sioux Falls, S. D.	Roentgenology
440.	Newcomet, William S.	Philadelphia, Pa.	Radiology
441.	Newell, Robert R.	San Francisco, Cal.	Radiology
442.	Nichols, Bernard H.	Cleveland, O.	Radiology
443.	Nichols, Harold E.	Seattle, Wash.	Diagnostic Roentgenology
444.	Nordin, Gustaf T.	Minneapolis, Minn.	Radiology
445.	O'Boyle, Cyril P.	Philadelphia, Pa.	Diagnostic Roentgenology
446.	O'Brien, Frederick W.	Boston, Mass.	Radiology
447.	O'Connell, Andrew E.	Worcester, Mass.	Roentgenology
448.	O'Neill, John R.	San Francisco, Cal.	Roentgenology
449.	Oechsli, Waldo R.	Olive View, Cal.	Diagnostic Roentgenology
450.	Ogden, Ralph T.	Hartford, Conn.	Radiology
451.	Olin, Harry	Chicago, Ill.	Roentgenology
452.	Orndoff, Benjamin H.	Chicago, Ill.	Radiology
453.	Orr, Clifford R.	Buffalo, N. Y.	Radiology
454.	Osmond, John D.	Cleveland, O.	Roentgenology
455.	Osmond, Leslie H.	Pittsburgh, Pa.	Radiology
456.	Ostrum, Herman W.	Philadelphia, Pa.	Roentgenology
457.	Ourian, Adom K.	New York, N. Y.	Diagnostic Roentgenology
458.	Overgaard, Anders P.	Omaha, Neb.	Radiology
459.	Owen, Arthur K.	Topeka, Kan.	Roentgenology
460.	Owen, Colin C.	San Bernardino, Cal.	Roentgenology
*461.	Paine, Robert	Memphis, Tenn.	Radiology
462.	Pallen, Conde deS.	Rochelle Park, N. J.	Therapeutic Radiology
463.	Palmer, Dorwin L.	Portland, Ore.	Radiology
464.	Pancoast, Henry K.	Philadelphia, Pa.	Radiology
465.	Parker, Carl H.	Pasadena, Cal.	Roentgenology
*466.	Parry, Leo D.	Easton, Pa.	Radiology
467.	Paul, Lester W.	Madison, Wis.	Diagnostic Roentgenology
468.	Pawling, Jesse R.	Watertown, N. Y.	Diagnostic Roentgenology
469.	Peden, Joseph C.	St. Louis, Mo.	Roentgenology
470.	Peirce, Carleton B.	Ann Arbor, Mich.	Radiology
471.	Pendergrass, E. P.	Philadelphia, Pa.	Radiology
472.	Pendergrass, Robert C.	Americus, Ga.	Roentgenology
473.	Percival, Milton F.	Philadelphia, Pa.	Radiology
474.	Perkins, Charles W.	Norwalk, Conn.	Roentgenology
475.	Perry, Gentz	Evanston, Ill.	Radiology
476.	Peters, Chester M.	Canton, O.	Radiology
477.	Peterson, Charles H.	Roanoke, Va.	Roentgenology
478.	Peterson, Vernon L.	Charleston, W. Va.	Radiology
479.	Pfahler, George E.	Philadelphia, Pa.	Radiology
480.	Phillips, Clyde C.	Charlotte, N. C.	Radiology
481.	Pierce, Harold J.	Terre Haute, Ind.	Radiology
482.	Pierson, John W.	Baltimore, Md.	Radiology
483.	Pindell, Merl L.	Los Angeles, Cal.	Diagnostic Roentgenology
484.	Pines, John A.	Orlando, Fla.	Therapeutic Roentgenology
485.	Pirie, Alexander H.	Montreal, Que.	Radiology
486.	Pitts, Thomas A.	Columbia, S. C.	Roentgenology
*487.	Podlasky, Harry B.	Milwaukee, Wis.	Roentgenology
488.	Pohle, E. A.	Madison, Wis.	Therapeutic Radiology
489.	Pomeranz, Maurice M.	New York, N. Y.	Radiology
490.	Pomeranz, Raphael	Newark, N. J.	Roentgenology
491.	Pomeroy, Lawrence A.	Cleveland, O.	Radium Therapy
492.	Porter, Horace W.	Jackson, Mich.	Radiology
493.	Portmann, U. V.	Cleveland, O.	Radiology
494.	Post, Joseph W.	Philadelphia, Pa.	Diagnostic Roentgenology
495.	Potter, Carlton F.	Syracuse, N. Y.	Roentgenology
496.	Potter, Hollis E.	Chicago, Ill.	Roentgenology
497.	Potter, Roy P.	Marshfield, Wis.	Roentgenology
498.	Pound, Robert E.	New York, N. Y.	Roentgenology
499.	Powers, Martin T.	Utica, N. Y.	Diagnostic Roentgenology
500.	Powers, Robert A.	Palo Alto, Cal.	Radiology
501.	Prouty, J. V.	Terre Haute, Ind.	Radiology
502.	Putts, B. Swayne	Erie, Pa.	Roentgenology
503.	Quick, Douglas	New York, N. Y.	Therapeutic Radiology
504.	Quimby, A Judson	New York, N. Y.	Roentgenology
505.	Quiney, James J.	Easton, Pa.	Radiology
506.	Quinlan, Catherine M.	Santa Rosa, Cal.	Roentgenology
507.	Raap, Gerard	Miami, Fla.	Radiology

* Deceased.

508.	Radding, Moses B.	Elizabeth, N. J.	Diagnostic Roentgenology
509.	Rathbone, Ralph R.	Washington, D. C.	Radiology
510.	Rauschenbach, Charles W.	Hammond, Ind.	Roentgenology
511.	Ravold, Henry J.	St. Joseph, Mo.	Radiology
512.	Ray, William B. G.	Pittsburgh, Pa.	Roentgenology
513.	Reaves, Hugh G.	Knoxville, Tenn.	Roentgenology
514.	Reed, Charles B.	Newburgh, N. Y.	Roentgenology
515.	Reeves, Robert J.	Durham, N. C.	Radiology
516.	Reiley, William E.	Clearfield, Pa.	Radiology
517.	Reineke, Harold G.	Cincinnati, O.	Roentgenology
518.	Reitter, George S.	East Orange, N. J.	Radiology
519.	Remer, John	New York, N. Y.	Therapeutic Roentgenology
520.	Rendich, Richard A.	Brooklyn, N. Y.	Roentgenology
521.	Reynolds, Lawrence	Detroit, Mich.	Radiology
522.	Rhinehart, Darmon A.	Little Rock, Ark.	Roentgenology
523.	Rhudy, Booker E.	Greensboro, N. C.	Roentgenology
524.	Rice, Frank M.	San Francisco, Cal.	Roentgenology
525.	Rigler, Leo G.	Minneapolis, Minn.	Radiology
526.	Ritvo, Max	Boston, Mass.	Radiology
527.	Ritzman, Allen Z.	Harrisburg, Pa.	Roentgenology
528.	Robin, Nathaniel H.	Hempstead, N. Y.	Roentgenology
529.	Robin, Percival A.	Hempstead, N. Y.	Roentgenology
530.	Robins, Samuel A.	Boston, Mass.	Roentgenology
*531.	Robinson, Ralph V.	Pittsburgh, Pa.	Roentgenology
532.	Robinson, Walter W.	Memphis, Tenn.	Roentgenology
533.	Rodenbaugh, Frederick H.	San Francisco, Cal.	Radiology
534.	Rodgers, Floyd D.	Columbia, S. C.	Radiology
535.	Rodriguez, Juan	Fort Wayne, Ind.	Radiology
*536.	Roland, Marion M.	Oklahoma City, Okla.	Therapeutic Radiology
537.	Rose, Cassie B.	Chicago, Ill.	Radiology
538.	Rosenbaum, George	Philadelphia, Pa.	Diagnostic Roentgenology
539.	Rosh, Rieva	New York, N. Y.	Therapeutic Radiology
540.	Rousseau, J. P.	Winston Salem, N. C.	Radiology
541.	Rowe, Edward W.	Lincoln, Neb.	Radiology
542.	Rowell, Edward E.	Stamford, Conn.	Roentgenology
543.	Rubert, Samuel R.	Chicago. Ill.	Diagnostic Roentgenology
544.	Rudisill, Hillyer, Jr.	Charleston, S. C.	Radiology
545.	Ruggles, Howard E.	San Francisco, Cal.	Roentgenology
546.	Rutledge, Clifford P.	Shreveport, La.	Radiology
547.	Rypins, Edwin L.	Bloomington, Ill.	Radiology
548.	Sanderson, Stevens S.	Detroit, Mich.	Radiology
549.	Sante, L. R.	St. Louis, Mo.	Radiology
550.	Schimmelpfennig, R. D.	Montclair, N. J.	Roentgenology
551.	Schmitz, Henry	Chicago, Ill.	Therapeutic Radiology
552.	Schnoebelen, Paul C.	St. Louis, Mo.	Roentgenology
553.	Schons, Edward	St. Paul, Minn.	Radiology
554.	Schumacher, F. L.	Pittsburgh, Pa.	Radiology
555.	Schwartz, Charles W.	New York, N. Y.	Roentgenology
556.	Schwartz, Irving	New York, N. Y.	Diagnostic Roentgenology
557.	Sharpe, A. Maxwell	Chester, Pa.	Roentgenology
558.	Shaw, Wilfred McL.	Jacksonville, Fla.	Roentgenology
559.	Shebesta, Emil M.	Detroit, Mich.	Radiology
560.	Sheldon, Francis B.	Stockton, Cal.	Radiology
561.	Sheridan, William M.	Spartanburg, S. C.	Radiology
562.	Sherman, Herbert deG.	White Plains, N. Y.	Diagnostic Roentgenology
563.	Sherrick, Earl C.	Connellsville, Pa.	Diagnostic Roentgenology
564.	Shiflett, E. Lee	Indianapolis, Ind.	Roentgenology
565.	Shore, O. J.	Detroit, Mich.	Diagnostic Roentgenology
566.	Shoulders, H. S.	Nashville, Tenn.	Radiology
567.	Sichler, Harper G.	Lafayette, Ind.	Radiology
568.	Sickels, Thomas N.	Watertown, N. Y.	Roentgenology
569.	Siefert, Alfred C.	Oakland, Cal.	Radiology
570.	Sigman, Frederick G.	Salisbury, N. C.	Roentgenology
571.	Sims, George P.	Columbus, O.	Therapeutic Radiology
572.	Skinner, Edward H.	Kansas City, Mo.	Radiology
573.	Skomp, C. E.	Denver, Col.	Diagnostic Roentgenology
574.	Smith, Arthur B.	La Jolla, Cal.	Diagnostic Roentgenology
575.	Smith, Lester A.	Indianapolis, Ind.	Radiology
576.	Smith, Richard L.	Grand Rapids, Mich.	Radiology
577.	Smith, R. Manges	Philadelphia, Pa.	Roentgenology
578.	Smith, Roscoe L.	Lincoln, Neb.	Radiology
579.	Smyth, Thomas L.	Allentown, Pa.	Roentgenology
580.	Snead, Lawrence O.	Richmond, Va.	Roentgenology
581.	Snow, Henry	Dayton, O.	Radiology
582.	Snow, William	New York, N. Y.	Roentgenology

* Deceased.

583.	Snure, Henry	Los Angeles, Cal.	Roentgenology
584.	Soiland, Albert	Los Angeles, Cal.	Radiology
585.	Solis-Cohen, Leon	Philadelphia, Pa.	Radiology
586.	Sosman, M. C.	Boston, Mass.	Radiology
587.	Soule, Arthur B., Jr.	Burlington, Vt.	Roentgenology
588.	Spackman, Edgar W.	Philadelphia, Pa.	Radiology
589.	Spangler, Davis	Dallas, Texas	Radiology
590.	Spencer, Hunter B.	Lynchburg, Va.	Roentgenology
591.	Spilman, Harold A.	Ottumwa, Ia.	Roentgenology
592.	Spinzig, Edgar W.	St. Louis, Mo.	Radiology
593.	Sproull, John	Haverhill, Mass.	Roentgenology
594.	Stafford, Owen R.	Los Angeles, Cal.	Therapeutic Roentgenology
595.	Stall, Arthur H.	Akron, O.	Radiology
596.	Stammel, Charles A.	Washington, D. C.	Diagnostic Roentgenology
597.	Starks, Dorothy J.	Palo Alto, Cal.	Radiology
598.	Startz, Irving S.	Elmhurst, N. Y.	Roentgenology
599.	Stayton, Chester A.	Indianapolis, Ind.	Roentgenology
600.	Stecher, William R.	Darby, Pa.	Radiology
601.	Steel, David	Cleveland, O.	Roentgenology
602.	Steiner, Joseph M.	New York, N. Y.	Roentgenology
603.	Stephenson, F. B.	Denver, Col.	Radiology
604.	Stevens, Rollin H.	Detroit, Mich.	Radiology
605.	Stewart, Harry M.	Johnstown, Pa.	Radiology
606.	Stewart, Wendell	East St. Louis, Ill.	Diagnostic Roentgenology
607.	Stewart, William H.	New York, N. Y.	Roentgenology
608.	Stone, Robert S.	San Francisco, Cal.	Roentgenology
609.	Stuart, Leon H.	Tulsa, Okla.	Roentgenology
610.	Sutherland, Charles G.	Rochester, Minn.	Diagnostic Roentgenology
611.	Swenson, Paul C.	New York, N. Y.	Roentgenology
612.	Swope, Opie W.	Wichita, Kansas	Radiology
613.	Tabb, John L., Jr.	Richmond, Va.	Roentgenology
614.	Taft, Robert B.	Charleston, S. C.	Radiology
615.	Talley, Daniel D., Jr.	Richmond, Va.	Roentgenology
616.	Tamarkin, Saul J.	Youngstown, O.	Radiology
617.	Taormina, Louis J.	Brooklyn, N. Y.	Diagnostic Roentgenology
618.	Taylor, Clifford C.	Indianapolis, Ind.	Radiology
619.	Taylor, Henry K.	New York, N. Y.	Diagnostic Roentgenology
620.	Taylor, Raymond G.	Los Angeles, Cal.	Radiology
621.	Taylor, Richard T.	Los Angeles, Cal.	Radiology
622.	Teitelbaum, Meyer D.	New Orleans, La.	Roentgenology
623.	Thomas, Merthyn A.	Cleveland, O.	Radiology
624.	Thompson, Harold B.	Seattle, Wash.	Radiology
625.	Thuresson, Paul F.	Riverside, Cal.	Roentgenology
626.	Tice, Galen M.	Kansas City, Kan.	Radiology
627.	Titterington, Paul F.	St. Louis, Mo.	Roentgenology
628.	Troje, Oscar R.	Fairfield, Ala.	Diagnostic Roentgenology
629.	Trostler, Isador S.	Chicago, Ill.	Radiology
630.	Troup, Ralph L.	Green Bay, Wis.	Radiology
631.	Troxell, William C.	Allentown, Pa.	Roentgenology
632.	Tyler, Albert F.	Omaha, Neb.	Radiology
633.	Ude, Walter H.	Minneapolis, Minn.	Roentgenology
634.	Ulbrich, Henry L.	Detroit, Mich.	Roentgenology
635.	Ullmann, Henry J.	Santa Barbara, Cal.	Radiology
636.	Unger, Arthur S.	New York, N. Y.	Roentgenology
637.	Upson, Wilbur O.	Battle Creek, Mich.	Radiology
638.	Van Allen, Harvey W.	Springfield, Mass.	Radiology
639.	Van Buskirk, Edmund M.	Fort Wayne, Ind.	Roentgenology
640.	Van Nuys, Roscoe G.	Berkeley, Cal.	Radiology
641.	Vastine, Jacob H.	Philadelphia, Pa.	Radiology
642.	Velkoff, Metodi	Fort Wayne, Ind.	Roentgenology
643.	Virden, C. Edgar	Kansas City, Mo.	Radiology
644.	Vogt, Edward C.	Boston, Mass.	Roentgenology
645.	Voke, Edward L.	Akron, O.	Diagnostic Roentgenology
646.	Voltz, Albert L.	Richmond Hill, N. Y.	Roentgenology
647.	von Poswick, Gisela	Scranton, Pa.	Roentgenology
648.	Wachowski, T. J.	Chicago, Ill.	Roentgenology
649.	Walton, Henry J.	Baltimore, Md.	Radiology
650.	Warfield, Chester H.	Chicago, Ill.	Roentgenology
651.	Warren, Alva H.	Malden, Mass.	Diagnostic Roentgenology
652.	Wasson, W. W.	Denver, Col.	Radiology
653.	Waters, Charles A.	Baltimore, Md.	Radiology
654.	Watkins, W. Warner	Phoenix, Ariz.	Radiology
655.	Weaver, Clarence E.	Detroit, Mich.	Roentgenology
656.	Webb, James A. H.	Wichita, Kansas	Roentgenology
657.	Weber, Harry M.	Rochester, Minn.	Roentgenology
658.	Weeks, Paul R.	Denver, Col.	Radiology

659.	Weiskotten, W. Otto	San Diego, Cal.	Roentgenology
660.	Weitzner, Imre	New York, N. Y.	Diagnostic Roentgenology
661.	Weitzner, Samuel F.	New York, N. Y.	Diagnostic Roentgenology
662.	Wescott, William C.	Atlantic City, N. J.	Radiology
663.	West, James H.	Cleveland, O.	Radiology
664.	West, Theodore S.	Port Chester, N. Y.	Roentgenology
665.	Wheatley, Frank E.	Boston, Mass.	Radiology
666.	Wheatley, Louis F.	New Haven, Conn.	Roentgenology
667.	Whelan, Charles	Boston, Mass.	Radiology
668.	Whitehead, Lawther J.	Richmond, Va.	Roentgenology
669.	Whitlock, Simon B.	Norfolk, Va.	Roentgenology
670.	Widmann, Bernard P.	Philadelphia, Pa.	Radiology
671.	Wigby, Palmer E.	Dallas, Texas	Radiology
672.	Wilcox, Clark A.	Wichita Falls, Texas	Radiology
673.	Williams, Arthur J.	San Francisco, Cal.	Radiology
674.	Williams, Francis H.	Boston, Mass.	Radiology
675.	Williams, Lester J.	Baton Rouge, La.	Radiology
676.	Williams, Percival A.	Hempstead, N. Y.	Diagnostic Roentgenology
677.	Willy, Ralph G.	Chicago, Ill.	Roentgenology
678.	Wilson, Hugh M.	New Haven, Conn.	Roentgenology
679.	Wilson, James E.	Clarksburg, W. Va.	Diagnostic Roentgenology
680.	Wilson, Rabun T.	Temple, Texas	Roentgenology
681.	Wilson, Stanley A.	Lewiston, Me.	Roentgenology
682.	Withers, Sanford M.	Denver, Col.	Therapeutic Radiology
683.	Witwer, Eldwin R.	Detroit, Mich.	Radiology
684.	Woolford, Joseph S.	Eureka, Cal.	Therapeutic Radiology
685.	Wright, Cecil S.	Indianapolis, Ind.	Radiology
686.	Wurster, Lloyd E.	Williamsport, Pa.	Roentgenology
687.	Young, Barton R.	Philadelphia, Pa.	Radiology
688.	Zink, Oscar C.	St. Louis, Mo.	Radiology
689.	Zulick, J. Donald	Philadelphia, Pa.	Roentgenology

There will be two meetings of the Board in 1936 for the purpose of conducting examinations, one in Kansas City, Missouri, in May, just preceding the meeting of the American Medical Association, and the other in Cleveland, Ohio, in September, preceding the meeting of the American Roentgen Ray Society. Any applicant wishing to be assured of an appointment to appear before the Board in Kansas City should have his application on file in this office not later than March 1, 1936.

B. R. KIRKLIN, M.D.
Secretary-Treasurer,
The American Board of Radiology

COMMUNICATIONS

LOS ANGELES COUNTY MEDICAL ASSOCIATION, RADIOLOGICAL SECTION

The officers for the Radiological Section of the Los Angeles County Medical Association for 1936 are as follows: *President,* J. W. Crossan, M.D.; *Vice-President,* D. R. MacColl, M.D.; *Treasurer,* Henry Snure, M.D., and *Secretary,* E. N. Liljedahl, M.D.

Meetings are held every second Wednesday of the month at the County Society Building.

MINNESOTA RADIOLOGICAL SOCIETY

The Minnesota Radiological Society held its winter meeting at the Nicollet Hotel, in Minneapolis, Saturday, December 14. The following program was presented:

Radiation Therapy of Carcinoma of the Lip, by A. L. Abraham, M.D., and W. K. Stenstrom, Ph.D., Minneapolis.

Roentgen Study of the Results of Epiphysial Injuries, by Oscar Lipschultz, M.D., Minneapolis.

The Dangers of Reducing Fractures under the Fluoroscope, by E. T. Leddy, M.D., and C. A. Stevenson, M.D., Rochester.

Demonstration of Some Bone Changes in Children, by M. J. Shapiro M.D., Minneapolis.

Roentgen Study of Spontaneous Internal Biliary Fistula, by C. N. Borman, M.D., Minneapolis.

Experiences with Roentgen Therapy of More than 3,000 r in One Month, by W. K. Stenstrom, Ph.D., and Lewis Jacobs, M.D., Minneapolis.

Combined Heat and Roentgen Therapy of Tumors: Review of the Work of Stafford Warren and Associates, by C. O. Hansen, M.D., Minot, N. D.

The next meeting of the Society will be held in St. Paul in March.

Photograph by Watton Studio

The late MARION M. ROLAND, M.D.

IN MEMORIAM

MARION M. ROLAND, M.D.

Dr. Marion M. Roland was born near Bowie, Texas, in 1882, and died on a Sante Fe train Dec. 1, 1935, of coronary thrombosis while on his way to Detroit to attend the Annual Meeting of the Radiological Society of North America. He leaves his wife, Hazel I. Roland; one child, Marion M. Roland, Jr., D.D.S.; a brother, George E. Roland, D.D.S., and his aged mother, all living in Oklahoma City.

Dr. Roland came to Oklahoma with his parents in 1899 and settled on a farm near Weatherford, where he grew to manhood and married Hazel Nichols, daughter of a prominent druggist of Weatherford.

He attended Vanderbilt University three years, then transferred to Jefferson Medical College where he was graduated in 1908. After graduation he did a general practice at Eakley, Oklahoma, then joined in partnership with Dr. Everett S. Lain of Oklahoma City, Jan. 1, 1910. This congenial, happy partnership continued until his death.

Dr. Roland was a volunteer during the early months of the World War, and served as a radiologist for 22 months—11 months overseas.

He was a Fellow of the American Medical Association, member of the Southern Medical Association, the Radiological Society of North America, and had recently been honored with a fellowship in the American College of Radiology. He was an associate professor in the University of Oklahoma School of Medicine. His knowledge and skill in therapeutic radiology were unexcelled anywhere in the Southwest.

He was a member of the Baptist Church, Oklahoma City Rotary Club, Chamber of Commerce, the Shrine, Knight Templars, and Scottish Rites Masonry.

Dr. Roland was widely known for his congenial, cheerful, optimistic personality which deeply endeared him not only to all his office associates but to the multitudes with whom he came in contact during his professional life. His heart was as tender as a child's. He was generous almost to a fault with financial and other assistance to friends, medical students and even to the trudging peddler on the street.

Our medical profession has lost an outstanding radiologist and valued member, his family a devoted, loving husband, father, and brother, and the Lain-Roland-Eastland Clinic a kindly and beloved fellow associate.

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ANDREW H. DOWDY, M.D., of Detroit, Mich.
J. E. HABBE, M.D., of Milwaukee, Wis.
HANS W. HEFKE, M.D., of Milwaukee, Wis.
H. A. JARRE, M.D., of Detroit, Mich.
DAVIS H. PARDOLL, M.D., of Chicago
E. A. POHLE, M.D., Ph.D., of Madison, Wis.
EMIL M. SHEBESTA, M.D., of Detroit, Mich.

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ADDISON'S DISEASE

Calcifications of the Adrenals. A. Beutel. Röntgenpraxis, February, 1935, **7,** 85–87.

The roentgen examination of the adrenals in all cases of suspected Addison's disease is necessary for a complete diagnosis. The finding of bilateral calcification in the adrenals, with clinical symptoms, means tuberculosis of the adrenals. Occasionally only one side is found to show calcium deposits.

HANS W. HEFKE, M.D.

CANCER (THERAPY)

Factors Influencing the Determination of Radiosensitivity of Cancers of the Oral Cavity and Upper Respiratory Tracts. Arthur Purdy Stout. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 327–331.

An estimation of radiosensitivity of oral, nasal, and laryngeal neoplasms necessitates the consideration of numerous factors both extrinsic and intrinsic. This investigation has shown that infection, invasion of bone and cartilage, deep infiltration, and syphilis tend to make this group of cancers statistically somewhat more resistant than the absence of these features. Small size and the presence of leukoplakia favored statistical radiosensitivity. There was marked variation according to anatomical site; vocal cord, nasal, antral, cheek, and gum tumors included a high percentage of resistant tumors, while extrinsic laryngeal, nasopharyngeal, tongue, floor of mouth, and lower lip tumors included a high percentage of sensitive neoplasms.

The microscopic features which seemed to have the greatest significance were: (*a*) *As indications of resistance;* secretion of mucin, nerve sheath invasion, calcification, formation of glands, melanin, collagen fibers (in the fibrosarcoma), and other adult structures; and, in the squamous-cell epitheliomas, the presence of much keratin, many intercellular bridges, and many pearls. (*b*) *As indications of sensitivity;* lymphoepitheliomas, lymphosarcomas and, in the squamous-cell epitheliomas, the complete or relative absence of keratin, pearls, and intercellular bridges.

The features studied which seemed to have little or no importance as indicators of relative radiosensitivity were: the presence or absence of metastases, papillary tumors, bizarre cells, and mitoses. The presence or absence of stratification is too difficult to recognize to warrant its use as an indicator.

S. M. ATKINS, M.D.

Histologic Structure of Carcinoma of the Cervix Uteri and its Relation to Radiosensitivity. Charles C. Norris. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 332–337.

All cervical carcinomas are quite radiosensitive. As compared to many other tumors, the difference in sensitivity between the most radiosensitive types of carcinoma of the cervix and the most radioresistant is relatively slight. The fact that the final salvage is small is due not to radioresistance on the part of the tumor, but to the difficulty of securing intensive diffuse irradiation of the entire growth, including the metastases.

As compared to many other tumors, all cervical carcinomas are relatively highly malignant. In them, the radiosensitivity and the malignancy index tend to run more or less parallel. The greater malignancy probably counterbalances the greater radiosensitivity and in part accounts for the relatively slight differences in the five-year salvage of the different types.

As the normal cervical tissue is capable of withstanding extremely intensive irradiation, and taking into account the relatively high malignancy of all cervical carcinomas, our aim should be to apply as intensive and as diffuse irradiation as possible to all except definitely hopeless cases.

Histologic examination is neither a very practical nor a reliable guide as to either the prognosis or the radiation dosage. It may be of some use in groups, but means little in the individual case.

Papillary tumors are generally more radiosensitive than are those of the infiltrating type.

S. M. ATKINS, M.D.

Three and One-half Years of Roentgen Therapy with from 600 to 900 Kilovolts. S. G. Mudd and C. K. Emery. Strahlentherapie, 1935, **54,** 216.

This paper was presented before the Fourth International Congress on Radiology in Zürich. It offers a brief description of the apparatus used in the work and gives a report of 38 cases of carcinoma of the rectum and 33 cases of carcinoma of the prostate treated during the past three and one-half years. No definite conclusions are offered. (See also Am. Jour. Roentgenol. and Rad. Ther., 1934, **31,** 520.)

ERNST A. POHLE, M.D., Ph.D.

Radiation Therapy in Carcinoma of the Cervix. B. Guedes. Strahlentherapie, 1935, **54,** 238.

At the Institute of Cancer Research, in Lissabon, 299 cases of carcinoma of the cervix were treated by irradiation during the period from 1920 to 1928. Reexamination and statistical analysis of these patients in December, 1933, showed a 5-year cure of 21.7 per cent. Four tables are appended, giving all necessary data for the evaluation of the results. Histologic studies undertaken on 289 patients revealed that basal-cell carcinoma responded best to irradiation. Treatment technic: In the first few years large amounts of radium were left in the cervix for a short time; later on, the amount of radium was reduced to from 37.5 to 62.5 mg., for from 96 to 144 hours. (Filter 0.5 mm. Au and 1–1.5 mm. Pb.) The total dose applied varied between 4,000 and 8,000 milligram-hours. Eight days after the radium treatment the patient receives diathermy.

Beneficial results have been reported by the director of the Institute in a recent publication (Bull. Assn. Franç. Étude Cancer, 1930, **19,** No. 6) and consequently

this method has been made a routine procedure. Beginning in 1927, all cases received roentgen therapy before the radium application. Two abdominal and two posterior fields of 10 × 15 cm. are given, 600–900 r per field, and a total dose of 2,400 to 3,600 r in from 8 to 12 days (156–180 kv., 0.5–1.0 mm. Cu, 30–40 cm. F.S.D.).

ERNST A. POHLE, M.D., Ph.D.

HEART AND VASCULAR SYSTEM

Roentgen Diagnosis of Aneurysms of the Innominate Artery. Chester H. Warfield. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 350–358.

In the last six years the author has studied 20 cases of this type, 12 of which came to autopsy. The roentgen findings demonstrate that aortitis is always present preceding the aneurysm. The innominate aneurysm appears as a superior mediastinal tumor on the right side at the upper level of the arch of the aorta and extends above the inner end of the clavicle. In very large types it will extend to the left side also. In the lateral and oblique views, it is seen bulging posteriorly and compressing the trachea. This latter finding was constant. Deviation of the trachea to the left was present in only 15 per cent, since the innominate artery lies anterior to the trachea rather than to the right. This compression of the trachea frequently produces decubitus ulcers.

Clinically, there are no pathognomonic signs. The history of this condition, its anatomy, pathology, clinical findings, and differential diagnosis, are discussed.

S. M. ATKINS, M.D.

THE HIP JOINT

Congenital Coxa Vara. Isadore Zadek. Arch. Surg., January, 1935, **30,** 62–102.

The author presents a complete review of the literature and presents 10 cases of his own. Eight of the patients were children and two were adults. Four were males and six were females. Three of the children were colored. The two adults were white. There was bilateral involvement in four cases. The etiology of the condition is not known. The author summarizes various theories. The microscopic picture of tissue removed from the affected portion of the neck reveals nothing characteristic.

The history and physical findings are important. Deformity and a painless limp are noted in childhood. Pain begins in the adolescent or adult life if the patient remains untreated. If the neck of the femur has given way with resulting non-union, the disability is marked. Examination reveals elevation of the greater trochanter, limitation of abduction, sometimes also limitation of internal rotation, and more rarely, limitation of external rotation; a positive Trendelenburg's sign, and the head of the femur is palpable beneath the femoral artery in the groin. If the condition is bilateral, there is a marked increase in the lumbar lordosis. In contrast to congenital dislocation of the hip, the head of the femur is found in a more or less normal position with relation to Poupart's ligament, and when force is applied in the axis of the femur and relaxed alternately, the hip does not "pump" or "telescope." Unless there are some secondary changes around the hip joint, there is no muscle spasm and no tenderness.

Roentgen examination reveals a depression of the neck of the femur. There seems to be loss of substance in the neck which may be mistaken for a fracture with resulting non-union. The epiphyseal line is vertical with a branching, usually at its inferior portion. However, it may be at the superior margin. The greater trochanter is elevated and may even be in contact with the ilium. In the adolescent the defect in the neck of the femur may give way completely and a resulting non-union may be truly present, with no contact between the head fragment and the contiguous portion of the neck of the femir. On the other hand, there may be fibrous union. The head of the femur occupies the lower portion of the acetabulum and is often subluxated. At times the head is much larger than the attenuated neck, and has been described by Mayer as mushroom-shaped, appearing like the head of a giant on the neck of a dwarf. There may be considerable change in the acetabular outline. It is shallow and defective in its inferior portion and at times shadows are seen in the acetabulum which suggest osteochondritis.

Without surgical intervention the condition tends to become progressively worse, and in untreated patients to result in separation of the head of the femur and non-union. As a whole, the operative results are satisfactory. However, the prognosis is governed by the treatment. A conservative treatment is considered by the author to be obsolete. He considers the proper procedure to consist of two surgical phases: (1) Bony union should be produced in the defective portion of the neck of the femur; and (2) when this union is secured, the depressed neck of the femur should be elevated by a subtrochanteric wedge osteotomy.

ANDREW H. DOWDY, M.D.

THE KIDNEYS

Pre-operative Irradiation of Massive Tumors of the Kidney: A Clinical and Pathologic Study. Lawrence R. Wharton. Arch. Surg., January, 1935, **30,** 35–51.

The author states that, in general, of all patients who, on presentation, have had renal tumors, from 5 to 10 per cent have been refused operative treatment because of metastasis or the extent of the local growth. From 12 to 26 per cent have had only exploratory operations; from 15 to 30 per cent have died of operative shock, and, of the remainder, less than 10 per cent have been permanently cured. He feels that the greatest factor responsible for operative death is the size of the tumor. This factor has remained beyond control until the present time. Now, however, with the use of roentgen therapy pre-operatively in these cases, this handicap has been markedly overcome. In the author's experience, pre-operative irradiation has markedly reduced the size of the renal tumor and such shrinkage has usually been accomplished within a period of three weeks'

time. Of the cases reported in this article, two presented typical Grawitz's hypernephromas; one case was a girl two and one-half years of age and the other was a woman of 51. The other two growths were Wilms' embryomas in children 3 and 16 years of age, respectively.

Irradiation does not destroy all the tumor tissue. It does, however, in addition to producing marked shrinkage in the size of the tumor, kill and destroy a large number of malignant cells. These two factors diminish the likelihood of recurrences. The reduction in size makes the tumor more accessible and the operation can be done in a more cleanly manner, as the smaller tumor can be removed with little manipulation or bleeding. The surgeon is able to locate the renal pedicle before disturbing the tumor if the operation is done transperitoneally. The author emphasizes the fact that irradiation is an adjunct in the operative preparation and that surgery must be carried out at an appropriate time if any assurance of cure is to be offered. The patient must be studied carefully. It may be necessary to administer transfusions even before the irradiation. The white cell count is accurately followed and if there is significant reduction, or if there is definite leukopenia, the period of irradiations must be interrupted until the blood picture sufficiently approaches normal to warrant the completion of the treatment. He advocates the transperitoneal approach as being the preferable operation for nephrectomy in these cases.

The author presents a series of seven malignant renal tumors which are divided into three groups: (1) Large tumors in which nephrectomy was performed or attempted without pre-operative irradiation, (2) large tumors which were removed after pre-operative irradiation, and (3) small tumors which were removed without irradiation.

ANDREW H. DOWDY, M.D.

THE KNEE JOINT

Fossa intercondyloidea femoris and its Significance in Roentgenography of the Knee Joint. R. Kaiser. Bruns' Beitr. z. klin. Chir., June 5, 1935, **161,** 528–538. (Reprinted by permission from British Med. Jour., Sept. 21, 1935, p. 45 of Epitome of Current Medical Literature.)

The author regrets that Frik's method of eccentric radiography of the fossa intercondyloidea of the femur—described in 1932—has not found a wider recognition and use. He illustrates seven cases in which foreign bodies, loose bodies, fractures, or arthritic bony changes in the fossa were not shown, or were imperfectly shown, in the ordinary anteroposterior or lateral radiograms, but were clearly demonstrated lying within the roomy, well-defined notch which is pictured by Frik's technic. In this the central rays are impinged perpendicularly on the long axis of the notch: with the knee bent at an angle of from 125 to 130 degrees the tube is directed at right-angles to the tibial axis toward the lateral epicondyle, and then tilted slightly so as to illuminate the center of the joint cavity, immediately below the patella. In the resulting film the notch is seen unobscured by the shadows of the knee-cap and femoral condyles.

THE LIVER

The Chloride Content in the Tissues of the Rat Following Total Body Exposure to Roentgen Rays. Alois Beutel and K. Anton Winter. Strahlentherapie, 1935, **54,** 89–95.

The entire body of rats was exposed to roentgen rays (180 kv., 0.5 mm. Cu + 1.0 mm. Al, 4 ma., 40 cm. F.S.D., 750–1,000 r). An analysis of the organs and tissues showed that the chlorides were increased following irradiation regardless of whether the animals were examined on the first or third, second, fifth, or eighth day following the exposure. In all rats the chloride content of the liver was definitely decreased.

ERNST A. POHLE, M.D., Ph.D.

THE LUNGS

Fatal Bronchopneumonia Caused by Poisoning with Nitrous Gases. H. Beining. Röntgenpraxis, August, 1935, **7,** 534–541.

The literature on roentgenographic lung changes after inhalation of nitrous gasesis reviewed (only Nichols and Doub have contributed such studies). In the case seen by the author a man inhaled, apparently, very little nitric acid smoke. Cough and bloody sputum were noticed on the same day, but the patient felt well enough in a few days to go to work again. Eighteen days after the accident dyspnea and cough brought him back for examination. A roentgenogram at that time showed a normal heart; both lungs showed multiple miliary soft areas of increased density scattered throughout, similar to the picture of a miliary tuberculosis. An autopsy three days later showed a widespread bronchiolitis and bronchopneumonia.

Roentgenograms of the lungs in such cases are of great importance, as they show the bronchiolitic lesions and their extent, often before clinical symptoms indicate the severity of the process.

HANS W. HEFKE, M.D.

OSSIFICATION OF CARTILAGE

Ossification (So-called "Calcification") of Normal Laryngeal Cartilages Mistaken for Foreign Body: A Correlation of Roentgen and Histologic Findings. W. Edward Chamberlain and Barton R. Young. Am. Jour. Roentgenol. and Rad. Ther., April, 1935, **33,** 441–450.

Ossification of the hyaline cartilages of the larynx (the thyroid, cricoid, and greater part of the arytenoids)

begins normally when skeletal growth is otherwise complete. This ossification presents an appearance to radiographic study which is indistinguishable from calcification occurring as a result of degenerative changes in various tissues. However, the microscopic study of these laryngeal cartilages shows true bone formation. Such study shows true bone in thyroid and cricoid cartilages but only osteoid tissue in the arytenoids.

When the radiographic study of the pre-vertebral tissues of the neck shows fairly complete ossification of the cartilages there is little likelihood of misinterpretation in cases of suspected foreign body in the hypopharynx. When, however, the film study shows isolated linear zones of calcific density in the dorsal extremities of the laminæ of the thyroid or cricoid a considerable difficulty is experienced in distinguishing these from certain foreign bodies, such as chicken bones.

In the writers' experience errors have been made resulting in unnecessary endoscopic examinations, with failure to demonstrate any foreign body. On the other hand, the reverse situation of overlooking a foreign body by considering it a normal structure has not occurred.

One suggestion to aid in differentiating foreign bodies from normal ossification by improving detail and producing significant changes of relationship is to make a film after the patient has inflated the hypopharynx and other communicating spaces by the maneuver of forced expiration against closed lips and pinched nostrils.

Fortunately about 98 per cent of foreign bodies in this region are below the lower border of the cricoid cartilage. Any opaque linear density in this region is almost certain to be a foreign body.

J. Edwin Habbe, M.D.

THE PANCREAS

So-called "Pseudo-cysts" of the Pancreas and Bursitis Omentalis: Anatomic and Roentgenologic Observations. F. Gerth. Fortschr. a. d. Geb. d. Röntgenstrahlen, 1935, **51,** 8–22.

The author presents two case reports of cystic encapsulations in the pancreatic area of man's-head size. Case 1, female, 50 years of age, died in diabetic coma. The presence of a left upper quadrant tumor had been known for three years (probably it had existed for as long as seven years). It contained necrotic pancreas, and had recently ruptured to the duodenojejunal flexure. The roentgen report was an extragastric tumor of the left upper abdomen, probably arising from the left hepatic lobe or pancreas. The left kidney and spleen showed normal contours. The gastric mucosal relief was compressed but not intrinsically altered.

Case 2. Male, 66 years of age, had an abdominal mass which had developed suddenly within two weeks. Ileus was suspected. The roentgen report was displacement and compression of the stomach by a large left upper quadrant tumor, which also displaced the jejunum and the colon. The suspected tumor site was the pancreas or left kidney. Operation consisted of drainage of a large cyst in the bursa omentalis, containing necrotic pancreas. The wound gradually healed. The patient, who was afflicted with cardiorenal disease and had previously suffered an apoplectic stroke, died three months later in an emaciated condition. No autopsy was obtained, nor any report of urinalysis and blood sugar.

The paper closes with a more detailed discussion of the pathologic developments in the two cases as deduced from the history, autoptic and operative observations. To roentgenologists the report is of interest, showing how, by careful procedures, the origin of the abdominal masses could be narrowed down to a rather few possibilities. Pyelography might have been employed in Case 1, but probably was contra-indicated in Case 2.

H. A. Jarre, M.D.

PROTECTION

The Dangers of Roentgenoscopy and Methods of Protection against Them (IV. A Detailed Consideration of the Doses Received by the Fingers of the Examiner). Earl I. L. Cilley, B. R. Kirklin, and Eugene T. Leddy. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 390–395.

Owing to flexion of the finger tips in palpation they receive about three times the dose of the rest of the hand. For this reason a palpatory technic which utilizes the smallest possible beam is the safest.

S. M. Atkins, M.D.

RADIUM

Treatment of Benign Hemorrhages with Radioactive Substances. H. Eymer. Zentralbl. f. Gynäk., May 25, 1935, **59,** 1202–1207. (Reprinted by permission from British Med. Jour., Aug. 17, 1935, p. 28 of Epitome of Current Medical Literature.)

The author prefers radium to x-rays in treatment of "non-malignant" uterine hemorrhage because he has found that it causes less troublesome symptoms of artificial menopause and is certain that it acts directly on the endometrium. He mentions 512 "metropathic cases"—excessive hemorrhage, in the absence of gross disease, mostly near or beyond the climacteric—and 149 cases of myoma, treated by intra-uterine application of radium (or mesothorium), with no deaths and only a dozen failures. Such treatment in properly chosen cases is, therefore, regarded as indispensable in gynecological therapeutics. In myoma its scope is limited: the cavum must be not too long, and of regular configuration: preliminary sounding is essential; uteri reaching the navel are unsuitable, as are also general submucous myomas. As a rule irradiation is preceded by curettage, but Eymer does not hesitate to omit this in myomatous patients whose menses, though excessive, are perfectly regular. In many cases curetting is followed by irradiation at the same sitting. Contraindications other than those mentioned are (1) suspicion

of local inflammation, and (2) pyrexia, however slight. The dosage is 2,000 to 3,000 mg.-hr., with a brass filter 1 mm. thick. Sero-purulent, pink, or brownish discharge usually follows, lasting some weeks. In those aged under 44 years Eymer prefers operation, and in those with hyperpiesis in whom curetting has eliminated suspicion of malignancy he is reluctant to undertake any treatment at all.

Dosage and Technic in the Treatment of Carcinoma of the Uterine Fundus with Radium. Harry H. Bowing and Robert E. Fricke. Am. Jour. Roentgenol. and Rad. Ther., January, 1935, **33,** 50–53.

The basic principle consists of intensive application by the broken dose method and the use of simple applicators, which are gamma-ray tubes containing 50 millicuries of radon, as the unit of treatment; this permits wide choice, so that treatment can be individualized. As the lesion cannot be accurately outlined, the purpose of the authors is adequate homogeneous irradiation of the uterine canal, including the cervix when involved, supported by vaginal irradiation. Radium element needles are employed in cases of metastasis to the vaginal wall, and surface packs are used if the inguinal lymph nodes are involved.

Treatments with roentgen rays are given following the course of radium treatments, especially in inoperable cases and in those in which the lesions are of a high grade of malignancy.

Results have been encouraging. The whole problem is mainly surgical, although when operation is contraindicated many lesions may be satisfactorily arrested or apparently "cured" by careful irradiation.

In inoperable conditions, palliation is usually secured by limited treatments, and is an inestimable boon to the unfortunate patients.

The exact dosage and method of application is given for the various grades.

S. M. Atkins, M.D.

Physical Factors in Interstitial Radium Therapy. Edith H. Quimby. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 306–316.

The physical factors of radium dosage are the same for both external and interstitial sources, namely, length, strength, and filtration of the individual sources, their grouping and their distance from the tissue cells it is desired to affect. The duration of the irradiation, the so-called "time factor," while not, strictly speaking, a physical factor, should also be considered.

Beeswax, the bleeching of butter, and the production of necrosis in the dorsal muscles of rabbits were used to produce data regarding various types of interstitial sources of radiation.

All dosage data from these experiments were expressed in terms of the same unit as that used in external irradiation, the threshold erythema dose.

Tubes 12 mm. long with a filter equivalent to 0.5 mm. of platinum and strengths such that the exposures varied between one and three hours, produced a threshold erythema at a distance of 1 cm. in tissue with an irradiation of 150 mc.-hr. For other distances a table was shown.

Tables are also given showing the effect on dose of change in filter and length of implants, and of variation in distance in tissue.

The doses delivered to certain points in the neighborhood of groups of implants of different lengths have been calculated. From these results a curve and a table have been developed, making possible the determination of the minimum tissue dose delivered in a given mass, from a specified irradiation, for any type of interstitial sources in general use at present.

S. M. Atkins, M.D.

Clinical Considerations Influencing Radium Dosage. Maurice Lenz and J. R. Freid. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 319–326.

Clinical factors influencing radium dosage may depend on the general state of health of the patient or on local conditions in the tumor and tumor bed. Radium dosage may have to be reduced in extreme old age, heart disease, alcoholism, diabetes, etc., or because of the anatomic location and accessibility of the growth or its proximity to important normal structures, poor vascularization and infection of the tumor and tumor bed.

Destruction of tumor bed in esophageal carcinoma may lead to mediastinitis. Proximity of the eye demands special precautions to prevent eye injury. Radionecrosis may easily follow in the treatment of the sole of the foot. In the base of the tongue, aspiration pneumonia or lung abscess may follow the mucous membrane reaction. When squamous cell epithelioma invades bone or cartilage, it usually cannot be arrested by radium therapy alone; when close to either, necrosis or infection of them may occur. In cases in which the involvement is not readily accessible, as tumor of the external auditory canal, rectum, and esophagus, the dosage cannot be evenly distributed. Regressive changes in the tumor bed such as post-operative scar or syphilis reduce the resistance of the normal tissue. This is also true from previous radium therapy. In the presence of infection intensive therapy may result in septicemia. The resistance of skin to infection following intensive therapy is reduced.

Many illustrative cases are described.

S. M. Atkins, M.D.

Physical Factors in Teleradium Therapy. Wilhelm Stenstrom. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 296–301.

I. *Filters.*—Two mm. of brass removes practically 100 per cent of the beta rays, 50 per cent of the soft gamma rays, and 8 per cent of the hard gamma. Additional filter reduces the intensity without increasing the penetrability a great deal. Two mm. of pure rubber stops the secondary radiation from the metal. With these filters, 1 cm. of tissue absorbs 5 per cent of the radiation.

II. *Distance.*—An appreciable improvement in

depth dose can be obtained by increasing the distance but not by increasing the filter. Unfortunately, the expense of using radium at 20 cm. distance or more is, as a rule, too high to be considered.

III. *Distribution of Radium in Applicator.*—In a radium pack, it is most economical to place the radium peripherally inside the pack.

IV. *Skin Erythema Dose.*—If a filter of 2 mm. of brass and 2 mm. of rubber is used, 160 mg.-hr. may be considered as the amount required to produce a threshold erythema when a point source is used at 1 cm. distance. At other distances, to procure the same skin reaction from a point source, the square of the distance is multiplied by 160. For a ring source there is added to the point source 40 mg.-hr. for 1 cm. diameter and for larger diameters 40 times the square of the diameter.

S. M. Atkins, M.D.

Physical Factors in Intracavity Radium Therapy. J. L. Weatherwax. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 302–505.

The radiologist should plan intracavity treatment of a lesion, keeping in mind the following factors: (1) location of lesion; (2) distance of applicator from the surface of the lesion; (3) filter employed; (4) size and shape of applicator; (5) penetration.

Experience has demonstrated that in the treatment of many intracavity lesions, it is impossible to deliver a lethal dose throughout the entire lesion with radium alone.

The lethal zone produced by radium can be extended by combining roentgen irradiation, and it would seem advisable to have the height of tissue reaction for both radium and roentgen irradiation occur at approximately the same time.

S. M. Atkins, M.D.

The Physical Determination of Radium Dosages. Otto Glasser. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 293–295.

No satisfactory dosage unit for curietherapy similar to the r (roentgen) in roentgen therapy has been generally accepted. A unit is suggested called the "eve," which is equivalent to 7.35 roentgens and is produced by 1 gram of radium element per hour at 1 cm. distance calculated by means of Eve's constant. This value was arrived at under identical conditions by various physicists, including the author.

S. M. Atkins, M.D.

ROENTGEN SICKNESS

Pathogenesis, Prophylaxis, and Therapy of Roentgen Sickness. (Second communication.) A. Jugenburg, L. H. Peretz, and R. S. Mostowa. Fortschr. a. d. Geb. d. Röntgenstrahlen, March, 1935, **51,** 280–290.

In a previous communication (same journal, Volume 49), the authors reported a correlation between occurrence and severity of roentgen sickness, and changes in the flora of the intestinal tract. They came to the conclusion that the cause of roentgen sickness was a resorption of products of disintegration of the intestinal vegetation induced by the influence of roentgen rays upon the bacteria, and possibly also the bacteriophage.

In this paper they report on experimentations in prevention of roentgen sickness by administration of resistant types of colon bacilli prior to and during roentgen irradiation. According to this report, the results are most gratifying during the roentgen therapeutic management of various types of disease, particularly in a surprisingly large number of duodenal ulcers subjected to x-radiation. Corresponding animal experimentation is also reported.

The last paragraph of this paper states that Professor Nemenow, Director of the State Institute for Roentgenology, Radiology, and Cancer Research from which this paper emanates, does not share the views of the authors concerning the changes in the intestinal vegetation following roentgen irradiation. He suspects that changes in the intestinal flora represent a secondary manifestation induced by initial irritation of the vago-sympathetic balance with subsequent alteration of the chemistry of the intestinal tract, which, in turn, would produce subsequent bacterial changes. Such an interpretation would explain the occurrence of roentgen sickness during irradiation of extra-abdominal locations better than the view of the authors. While the paper seems to express an over-optimistic viewpoint and quite debatable theoretical conclusions, an experimental check-up would seem worth while.

H. A. Jarre, M.D.

THE SHOULDER JOINT

Roentgenographic Evidence in the Tuberosity of the Humerus of Recent and Old Injuries to the Supraspinatus Tendon Attachment. Lucas S. Henry. Am Jour. Roentgenol. and Rad. Ther., April, 1935, **33,** 486–490.

The author feels that many cases of undiagnosed shoulder disability following trauma are the result of supraspinatus tendon injury, which, both in the acute and chronic stages, may at times give roentgenologic evidence of its presence. Films using a soft tissue technic, taken with the arm adducted and externally rotated, may show, in recent injury cases of this nature, very fine spicules of thin bone or loosened periosteum projecting from the tuberosity. There is rapid atrophy or loss of density of the trabeculæ near the point of separation and then an appearance of irregular areas of increased density. In old cases the following three signs indicating rupture are: (1) atrophy of the trabeculæ beneath the base of the bursa so that the region of the tuberosity is more diaphanous; (2) irregular osteitis about the sulcus and the top of the tuberosity, and (3) the gradual disappearance of the sulcus in very old cases in which the tuberosity has been slowly absorbed.

J. E. Habbe, M.D.

THE SINUSES

To what Extent may Changes in Shape of the Sphenoid Sinus be Used for the Roentgen Diagnosis

of Endocranial Tumors? R. Mittermaier. Röntgenpraxis, August, 1935, **7,** 513–518.

Stenvers believes that changes in the shape of the sphenoid sinus may be used for differentiation between primary tumors of the pituitary and secondary widening of the sella turcica. The base of the sella remains parallel to the inferior wall of the sphenoid sinus when the widening is due to a primary tumor of the sella; when the widening is due to secondary causes (due to dilatation of the third ventricle), it takes place in an irregular fashion and anterior or posterior portions may be depressed independently. The author does not believe that such parallelism is necessarily found in normal individuals or in pathologic specimens. He admits the help of Stenvers' criteria but warns against using them indiscriminately in every case. Several cases are described to prove the author's point.

HANS W. HEFKE, M.D.

THE SPINE

Studies on the Sacrum: Cysts of the Sacrum. T. Bársony and Katharine Winkler. Röntgenpraxis, August, 1935, **7,** 505–512.

In Schmorl's series of pathologic studies of spines, cystic dilatation of the sacral canal was found in 1.16 per cent of 4,300 cases. Three such cases diagnosed roentgenologically *in vito* are described by the authors. The cystic cavities are usually bilateral and limited by a well-defined cortical layer. They are located (according to Kleiner's anatomical studies) most often about the second or third sacral segment. On lateral examination they are found to extend also anteriorly and posteriorly. The intervertebral disc is often narrowed. One must be careful not to mistake foramina sacralia or anomalies of the arch for cystic widening of the sacral canal. Clinical symptoms cannot be attributed to this anomaly.

HANS W. HEFKE, M.D.

Senile Osteomalacia: Report of a Case. Samuel Kleinberg. Arch. Surg., January, 1935, **30,** 30–34.

The author presents the case of a female 65 years of age with a two years' history of backache. This was not preceded by any known injury or illness. It was insidious in its onset, continually increasing in severity. There was a notable reduction in her height, with considerable kyphosis. The condition proceeded to total incapacitation. The patient was fairly well nourished for a person of her age.

Roentgenograms revealed severe atrophy of the vertebræ, most marked in the dorsal region with deformity of certain of the dorsal vertebræ. The seventh, ninth, and eleventh dorsal vertebræ were markedly reduced in their vertical diameters. The first lumbar vertebra was misshapen. Atrophy was also noted in the pelvic bones but the skull and extremities did not share in the pathologic change.

The author felt that malignancy could be ruled out due to the fact that there was no primary focus anywhere in the body, the lesion in the spine had extended over a period of two years without impairment of the general health, or cachexia, and the back pain was not as intense as that customarily experienced in a metastatic malignant process of the vertebræ. Hodgkin's disease was excluded. The blood calcium and phosphorus were of normal values. No neurologic disturbances could be elicited. The author concludes that the changes in the bone were on a nutritional or metabolic basis and that this nutritional upset might be considered a senescent degenerative change. The author was unable to explain the localization to a limited portion of the skeleton.

His treatment consisted of putting the patient to bed on a canvas frame. Gradually the frame was bent to a convex curve. As the curve in the frame was increased, the kyphosis of the spine was somewhat reduced. The patient was given, in addition, sedative doses of baking and massage to the back. Therapy was rapidly and surprisingly effective. Within a period of three weeks' time, the pain and tenderness disappeared, the kyphosis was reduced, and the patient, with the support of a light plaster of Paris jacket, was able to move about comfortably and was discharged.

ANDREW H. DOWDY, M.D.

Certain Aspects of Roentgenology of the Spine from the Orthopedic Viewpoint. Raymond W. Lewis. Am. Jour. Roentgenol. and Rad. Ther., April, 1935, **33,** 491–503.

As a means of testing for spinal muscle imbalance, two films are made, one with the patient sitting naturally in an anteroposterior position on a horizontal stool and the other with the hip on the convex side of the curve, raised about three inches by placing a book or some other suitable object under it. If in the second film with the hip elevated the curve can be straightened and the spine pulled into the vertical, there is evidence that the muscles on the side of suspected weakness are functioning well; if, on the other hand, the comparison fails to show this change, there is evidence of muscle imbalance.

To determine whether or not the deformed spine is flexible, films in appropriate position, with the patient in the supine and sitting positions, are made and the degrees of curvature compared. If the deformity is increased in the sitting position, the spine is obviously quite flexible.

It is the writer's opinion that in certain slight compression fractures of the vertebral body in which there are no breaks in the cortex and little or no disturbance of the structure of the body to be seen, it may be impossible to state with certainty whether the fracture is old or recent. He believes, in view of Baensch's demonstration of the amount of destruction of cancellous portions of the vertebral body which can escape roentgen detection as long as the cortex remains intact, that Kümmell's disease represents this type of bony injury which escapes immediate detection but subsequently develops a recognizable deformity.

It seems probable, according to the writer, that many cases of vertebral osteochondritis are being miscalled multiple herniations of the nucleus pulposus. These

abnormal appearances resulting from vertebral osteochondritis may also be erroneously diagnosed as arthritis, particularly in young adults.

Concerning osteomyelitis of the spine, great difficulty is encountered in differentiating it from tuberculosis: however, points in favor of osteomyelitis are: (1) rather advanced age of the patient; (2) pyogenic lesions occurring elsewhere in the body; (3) limited destruction of the vertebral bodies, and (4) early ankylosis due to bony proliferation about the lesion.

J. E. HABBE, M.D.

Unilateral Sacralization of the Entire Lumbar Spine. T. Rövekamp. Röntgenpraxis, August, 1935, **7,** 542, 543.

Sacralization of the fifth lumbar transverse process is seen in approximately 5 per cent of the cases. In this instance, however, the right transverse processes of the third, fourth, and fifth lumbar vertebræ formed wide bony plates, connected with each other by joints; and the first and second transverse processes were unusually large. Clinical symptoms were not present.

HANS W. HEFKE, M.D.

THE STOMACH

The Dangers of Roentgenoscopy and Methods of Protection against Them. III. The Protective Power of the Barium-filled Stomach. Earl I. L. Cilley, Eugene T. Leddy, and B. R. Kirklin. Am. Jour. Roentgenol. and Rad. Ther., January, 1935, **33,** 88–92.

The barium in the stomach during roentgenoscopic examination is an important protective element. Elicitation of the picture of the mucosal relief by deep palpation increases to a marked extent the dose of roentgen rays received by the examiner's hand. Data are presented to show that the emphasis placed by the roentgenoscopists of the Mayo Clinic on the value of the barium-filled stomach, and the smallest possible beam, as protective devices, is justified.

S. M. ATKINS, M.D.

Primary Isolated Lymphogranulomatosis of the Stomach: Report of a Case. Harry N. Comando. Arch. Surg., February, 1935, **30,** 228–235.

This article consists of the presentation of a case of localized Hodgkin's disease of the stomach which was operated on and has made an apparently satisfactory recovery.

The man had been complaining of a pain in the epigastrium for three years, beginning approximately three to four hours after meals. This was described as being of a dull character and not relieved by food. It radiated to the left side of the chest and to the back. Immediately after eating, his abdomen became distended. He belched a great deal and, on occasion, vomited. The vomitus consisted of food that had been eaten recently and never contained any blood. The stools were free of blood. The bowel movements were satisfactory. His appetite, general health, and strength were apparently good. He had lost 15 pounds in weight during the past year.

The physical examination was essentially negative. The Wassermann and Kahn tests were negative. The red blood cells were 4,550,000, with 80 per cent hemoglobin. The white blood cell count was 6,500, with 60 per cent polymorphonuclears, 37 per cent lymphocytes, 1 per cent eosinophils, and 1 per cent basophils. There was also reported 1 per cent endothelial cells. The urinalysis revealed a faint trace of albumin; otherwise it was negative.

The roentgen examination revealed changes which were those of an infiltration of the distal portion of the pars media and the proximal part of the pyloric end of the stomach. The roentgen diagnosis was infiltration of the stomach (providing syphilis was not present).

A clinical diagnosis of early carcinoma of the pylorus was made and a laparotomy was performed on May 6, 1930, under spinal anesthesia. A mass the size of a 25-cent piece was found on the lesser curvature of the stomach near the pylorus. This mass was pale, rather poorly defined, fairly hard, and definitely not inflammatory. Three or four glands the size of a small hazelnut were noted in the lesser omentum. A partial gastrectomy was performed, using Polya's method.

Gross examination of the specimen revealed the pylorus and the lower end of the stomach, measuring 10 cm. in length and 4 cm. in width. The serosa was smooth and glistening and showed no evidence of scars or any other pathologic condition. The wall of the stomach was soft and of normal thickness, except for an area over the lesser curvature about 4 cm. in diameter. This was firmer and felt thickened. On section through this area, the wall was seen to be markedly thickened, chiefly because of an infiltration in the submucosa. The muscularis, although it was slightly hypertrophied, did not appear to be involved in the process. The mucosa of the stomach was normal in appearance, except over the area already described, where definite atrophy and absence of rugæ were noted. A few ridges, which appeared to be infiltrated and were definitely indurated, were seen. There was no evidence of ulceration of the mucosa.

Microscopic examination of the sections revealed a diffuse infiltration by round cells with dark nuclei which resembled lymphoid cells. Under a high power lens many larger cells were seen, some of which were multinucleated and resembled typical cells of Reed and Sternberg. The glands of the mucosa appeared to be normal, and there was no evidence of epithelial neoplasia. The infiltration was confined to the submucosa and mucosa and did not extend to the muscularis in any of the sections that were studied. The pathologic diagnosis was Hodgkin's lymphogranuloma.

The patient's post-operative course was uneventful and he was discharged from the hospital on May 29. In a period of two months' time he had gained 25 pounds and had returned to his usual occupation, that of a helper on a farm. He had no dietary restrictions. A careful follow-up has shown no evidence of any recurrence and the patient continues to feel perfectly well.

The author arrived at the following conclusions: (1) Hodgkin's disease may originate in the lymphoid tissue

of the gastro-intestinal tract and remain as a localized tumor for some time. (2) The diagnosis usually made is that of carcinoma. (3) Before the days when gastric resection was the operation of choice for gastric tumors and for infiltrating gastric ulcers, practically no cases similar to this one were reported in the literature. (4) This type of gastric lesion is a distinct clinical entity, and the favorable results which have followed operative treatment in several cases more than justify the radical operation.

ANDREW H. DOWDY, M.D.

TUBERCULOSIS OF PHALANGES

Tuberculosis of the Phalanges in Older Individuals· B. Stenstrom. Acta Radiologica, 1935, **16,** 471–477.

The author describes three cases of tuberculosis of the phalanges occurring in individuals 29, 69, and 76, years old, respectively. X-ray examinations of these cases did not correspond to the usual picture of phalangeal tuberculosis—spina ventosa—commonly seen in children and young individuals. They resembled more osteomyelitis or infectious periostitis. Differential diagnosis is discussed, including chronic osteomyelitis, syphilis, tumors, and lymphogranuloma.

E. M. SHEBESTA, M.D.

TUMORS (DIAGNOSIS)

The Value of Roentgen Rays in the Diagnosis and Surgical Treatment of Extrapulmonary Intrathoracic Tumors. Stuart W. Harrington. Am. Jour. Roentgenol. and Rad. Ther., March, 1935, **33,** 340–349.

The clinical manifestations of these tumors are meager. The use of roentgen rays has made it possible to recognize them earlier, though many of them are difficult to solve even with the closest observation of the roentgenologist and clinician. Many of them require every method the roentgenologist can command, at times including pneumothorax, filling of the esophagus or the bronchial tree. Radiation therapy at times is of great aid, especially in ruling out mediastinal lymphoblastoma, which responds readily to radiation therapy.

Of the 47 cases reported, 19 were malignant and 28 benign. Fifteen were perineural fibroblastomas, of which two were malignant. Thirteen of these growths were in the posterior mediastinum. The benign showed sharp borders. Bone destruction in the benign, when present, was of the pressure type. In the malignant, there was a mortality of five.

S. M. ATKINS, M.D.

The Roentgen Diagnosis of Hypophyseal Tumors. Josef Erdelyi. Fortschr. a. d. Geb. d. Röntgenstrahlen, February, 1935, **51,** 125–147.

This paper is a detailed consideration of demonstrable changes in and near the sella produced by tumors of intrasellar origin—with and without acromegaly—of supra-, or infra-, para-, retro-, pra-sellar newgrowth and resulting from hydrocephalus. The normal variability of the sella is stressed, and it is mentioned that limited degrees of hypophyseal enlargement may not be accompanied by roentgenologic manifestations, even in the presence of distinct clinical symptoms of acromegaly or other signs of hypophyseal tumor. It is evident that the location of the original growth will often produce characteristic localizing roentgen changes, but when advanced destruction of the sella is present, when the dorsum is missing, and the sphenoidal sinus is completely compressed, the origin of the newgrowth may not be doubted any more. Remarks concerning hydrocephalus with herniation of the third ventricle into the sphenoidal sinus and illustrations of so-called multiple sellar contours, concentric in cases of hydrocephalus, converging in cases of asymmetrically growing tumors, are of interest. Ventriculography and encephalography are of minor importance in cases of tumors in or near the sella. Lipiodol is frowned upon because of its persistent confusing shadows.

Reading the paper and studying the 35 interesting illustrations one cannot help giving way to the impression that some of the deductions are drawn from inspection and autopsy; one might also wish for more information regarding the pathology of the material. This, however, should not prevent a careful review of this very worth while material selected from 80 cases of tumors in the sellar area.

H. A. JARRE, M.D.

TUMORS (THERAPY)

The Question of Supplementary Treatment of Malignant Tumors with Radio-active Bismuth. K. Overhof. Strahlentherapie, 1935, **54,** 261.

Experimentally it has been shown by Jansson that radio-active bismuth is deposited in greater amounts in the irradiated part of sarcomas in rats as compared with the unirradiated portions of the tumor. The author injected, therefore, intramuscular Bi-Diasporal (manufacturer: Klopfer, Dresden) in 125 cases of malignant disease. Ten cases were studied histologically; no bismuth could be shown histologically in liver, spleen, or tumor. Neither could its presence be detected chemically with a method sensitive enough to show 0.25 mg. of Bi-Diasporal. If used alone, no decrease in the tumor size was observed; if combined with radiation, it is difficult, of course, to decide how much benefit had been derived from the Bi-Diasporal medication and how much from irradiation alone. The author concludes, therefore, that further work is required before recommending the treatment of malignant tumors with radio-active bismuth.

ERNST A. POHLE, M.D., Ph.D.

Wilms' Tumor. William B. Coley. Am. Jour. Surg., September, 1935, **29,** 463, 464.

The author employs a toxin in inoperable case or in post-operative management of Wilms' tumor. It is his belief that surgeons will welcome the knowledge that they now have at their command a systemic agent which, in inoperable cases, also after nephrectomy in malignant kidney tumors, offers definite hope of lowering the appalling mortality. The article includes a

report on two cases in which the Coley toxin was employed.

DAVIS H. PARDOLL, M.D.

VENEREAL DISEASES

Cardiovascular Syphilis: Early Diagnosis and Clinical Course of Aortitis in 346 Cases of Syphilis. E. P. Maynard, Jr., J. A. Curran, I. T. Rosen, C. G. Williamson, and Claire Lingg. Archiv. Internal Med., June, 1935, **55**, 873–894.

This investigation studies the natural history of syphilitic infection in relation to its effect on the cardiovascular system. It was hoped that syphilitic involvement of the aorta could be detected earlier than is customary. The high incidence of syphilitic aortitis that was found seems to have justified this expectation, and emphasizes the necessity of periodic systematic examination, including the use of roentgenography and fluoroscopy, which provide the most reliable means of deciding whether abnormality of the aorta is present in early syphilis. Three hundred and forty-six patients with syphilis were studied, of which 41.9 per cent showed positive evidence of cardiovascular syphilis. In 14 adult patients with congenital syphilis no evidence of syphilis of the heart or aorta was found.

The date of occurrence of the primary lesion was known in about 55 per cent of the patients, with an average age of occurrence of 26 years. Evidence of cardiovascular syphilis was found within the first three years in eight patients (14 per cent), and one of these complained of symptoms referable to the heart. Evidence of cardiovascular syphilis was found within ten years after the appearance of the chancre in one-fourth of the cases, although the average interval from infection to the discovery of heart disease was twenty years. The average interval before symptoms occurred was twenty years.

Among 37 persons who were not examined until from 10 to 19 years after primary infection, 56.8 per cent exhibited evidence of cardiovascular syphilis. Symptoms appeared in 51.3 per cent of these. Heart failure occurred in two patients with aneurysm and aortic insufficiency.

In a group first observed from 20 to 29 years after infection, 77.8 per cent exhibited signs of syphilitic heart disease with symptoms present in 78 per cent. Of 17 persons who were examined 30 or more years after infection, 13 revealed the presence of cardiovascular syphilis, with 75 per cent complaining of symptoms. Heart failure occurred only in those patients with aortic insufficiency, aneurysm, or involvement of a coronary. Significant changes in electrocardiograms were present ten years after infection in only 10.4 per cent.

It is the opinion of the authors that involvement of the aorta begins soon after the chancre has appeared and that in the past discovery of the presence of the disease has been delayed by the late development of symptoms referable to the heart, and more especially by inadequate methods of examination.

EMIL M. SHEBESTA, M.D.

WANTED—Locum tenens to care for radiological practice with some general medicine during March and April, in central Wisconsin.

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